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FROM
THE LIBRARY
OF
SIR WILLIAM OSLER, BART.
OXFORD

3rd ed. 1898.

Freiburg i. Br.
Nov. 27th 1898

My dear Dr Osler,

I thank you very much for your letter of October 30th in which you announce to me the arrival of the 3d edition of your text-book from your publisher. The book has since arrived and, from what little I have had time to see by rapidly glancing over its contents, the opinion I have formed on its

Lundi, Mercredi, Vendredi,
de 3 à 5 heures.

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first appearance that it is the best text-book for completeness and conciseness of information I know of is fully borne out by this new edition.

I am very glad to see you so strongly go-in for the cold-water treatment of cases of typhoid and other fevers which in a text-book from authority and practitioners will have a very great effect in allaying the doubts existent every now and then by timid persons of limited experience as to the usefulness

or necessity of such
an treatment.

Allow me, on this occa-
sion, to allude to some
subjects, the elucidation
of which on a somewhat
wider base than they
are yours - upon so
far, seems to me some
importance. I mean
the questions regarding
the origin of arterio-
sclerosis and of cirrho-
sis of the liver. Both
subjects ought to be
further cleared - up,
in the first instance
as regards the influence
of race and of habits
of life on their pro-

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duction.

With regard to arteriosclero-
sis the question as to the in-
fluence infectious diseases
have on its origin ought
to be more carefully worked
out on a great material
in various countries.

The question as to the ca
of cirrhosis of the liver a
for as it depends on th
abuse of alcohol has go
a new shape since th
discussion raised by R
cereaux and the very in
portant communicatio
by L. Viola (Arch. gén.) on the or
currence of cirr. at
Venice and at Pad
from the use of plea
or non-pleasant dri

Perhaps your excellen

colleagues Professor Welch would take it in hand to gather exact information of the prevalence of the two diseases in various parts of the United States. If only all the large hospitals of this scientific work is done would for a time give carefully sifted records of the occurrence of these morbid conditions and their probable causes, much useful information might in time be collected.

The various points which have to be con-

98

de 3 à 5 heures. 179, Boulevard St Germain.

sidered in the vast question of extraterrestrial life I have tried to formulate in the papers of which I have sent you copies. But the question mentioned above as to the "spiritual" origin of Ast. seems to me one of the most important ones.

We were very glad your nephew Dr Gilliland's stay at Baltimore, and I wish you would oftener send us such nice young men over. If he is at Baltimore just

Now, will you, please,
give him mine and
my wife's compliments.

With sincere thanks,
for the presentation of your
book and with kind
regards

Yours,
very faithfully
C. Saint-Saëns.

[illegible]

, mon cher
ici de

ine,

heim

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Lundi, Mercredi, Vendredi
de 3 à 5 heures

179, Boulevard St Germain

Avec tous mes remerciements pour cet hommage de votre beau volume.

Docteur J. Dejerine,

Professeur Agrégé à la Faculté de Médecine

Médecin de la Salpêtrière

Paris le 28. 11. 93

Lundi, Mercredi, Vendredi

de 3 à 5 heures.

174, Boulevard St Germain

B.O. 3546

Tipped in volume 1:

1. On front pastedown, typescript letter addressed to Dr. Osler, from D. Appleton & Company, dated New York, November 12, 1898, with manuscript signature by R.B. G...[?];
2. On front endpaper, manuscript letter from Norman B. Allyn, dated June 14th 1901;
3. On front endpaper, a memo re B.O. 3546 in manuscript by W.W. Francis;
4. On title-page verso, clipping from Indiana Medical Journal, page 238, containing book review;
5. On page 10, manuscript letter addressed to Dr. Osler from George Brunner, dated Albany, N.Y., Jan. 12th 1901;
6. On page 17, manuscript letter addressed to Dr. Osler from Samuel A. Fish, dated Ashfield, Mass. Nov. 29, 1899;
7. On page 25, 3 leaves of typescript "Corrections", initialed by Dr. Osler, with 3 index cards in manuscript signed by W. Osler and rubber stamped "Publication Dept. Sep. 19, 1898".
8. On page 31, typescript letter to Dr. Osler from Dr. Aloysius O.J. Kelly, Philadelphia, dated November 10, 1900, with manuscript signature;
9. On page 31, an article entitled: The importance of the disinfection of the urine of typhoid fever patients, by The Department of Health of the City of New York;
10. On page 33, manuscript letter addressed to Dr. Osler from Charles P. Whitehead, dated Lecompte, La., 25 January 1901;
11. On page 33, manuscript letter addressed to Dr. Osler from E. Evans, dated Jan. 23rd, 1901;
12. On page 33, manuscript letter addressed to Dr. Osler from Dr. T. Caspar Gilchrist, Baltimore, MD, dated Feb. 1/01;
13. On page 33, typescript letter addressed to Dr. Osler from Dr. Chas. E. Rogers [Charles Elkins Robers], dated Montevideo, Minn., 1/31, 1901, with manuscript signature;
14. On page 35, article reprinted from the Boston Medical and Surgical Journal of January 12, 1899, entitled: An investigation as to the frequency of typhoid fever in infancy (4 pages);
15. On page 69, article reprinted from the Memphis Medical Monthly, June 1900, entitled: A warning in regard to vaccination: some comparisons by F.J. Runyon, M.D. (4 pages);
16. On page 69, manuscript letter addressed to Dr. Osler from Dr. F.J. Runyon, dated Clarksville, Tenn., June 15, 1900;
17. On page 74, manuscript letter addressed to Dr. Osler from Dr. W.C. Ussery, Paris, KY., dated April 7[?], 1900;
18. On page 77, clipping from the Boston Medical and Surgical Journal, vol. CXLII, no.24, June 14, 1900, pages 645-646, containing a correspondence entitled: Preliminary report on investigations in the bacteriology of Scarlet fever in the Scarlet-fever wards of the Children's Hospital, Berlin (Berlin, May 18, 1900) by W.F. Doolittle, M.D. of Cleveland, Ohio.
19. On page 84, manuscript letter to Dr. Osler from George W. Gay, M.D., dated Boston June 19, 1900;
20. Opposite page 90, manuscript annotations on slip re Rubella;
21. On page 138, 1 clipping of a chart indicating the deaths from Diphtheria in Chicago between 1886-1898; 1 pamphlet from Department of Health, City of Chicago; entitled: Diphtheria

Antitoxin treatment (8 pages); 3 leaves of typescript notes and tables concerning cases of Diphtheria treated with antitoxin by the Chicago Health Dept. between 1895-1900;

22. On page 146, laid in, manuscript letter addressed to Dr. Osler from Wm. H. Park, New York;
23. On page 153, pamphlet entitled: The hospital for Scarlet fever and Diphtheria patients, to the editor of The Medical Record, Edward L. Kellogg, M.D., New York (3 pages);
24. On page 287, clipping from the Albany Medical Annals, with a "Scientific Review" entitled: The relation of trauma to tuberculosis.
25. On page 338, laid in, typescript letter to Dr. Wm. Osler from Minnesota State Board of Health and Vital Statistics, H.M. Bracken [Henry Martyn Bracken], M.D., secretary, dated Dec. 11, 1900, with manuscript signature;
26. On page 338, manuscript letter to Dr. Osler, dated New Orleans ...[?] 3/1901, with illegible name of sender;
27. On page 349, an article reprinted from The Medical News, Mar. 4, 1899, entitled: A case of oestrus hominis by Maximilian Herzog, M.D. of Chicago, Ill.;
28. On page 370, note on card addressed to Dr. Osler from 531 Franklin St., Buffalo, with illegible name of sender;
29. On page 375, typescript letter to Dr. Osler from U.S. Dept. of Agriculture, Bureau of Animal Industry, Washington D.C., dated July 8, 1895, signed by D.E. Salmon [Daniel Elmer Salmon], Chief of Bureau;
30. On page 375, typescript letter from Dr. Osler, dated Baltimore, May 21st, 1895, signed in manuscript;
31. On page 375, manuscript letter to Dr. Osler from W.H. Evans, dated Sedalia, MO, July 19, 1895;
32. On page 386, manuscript letter to Dr. Osler from Headquarters Department of the East, Chief Surgeon's Office, Governor's Island, N.Y., dated March 21st, 1900, signed C.C. Byrne;
33. On page 402, 1 leaf of notes initialed by W.O.
34. On page 405, manuscript letter to Dr. Osler from Birmingham, dated 24 Sept., 1898, signed by Robert S...[?];
35. On page 406, typescript letter on onion skin to Mr. Steward Tupper, c/o Sir Charles Tupper, Ottawa, signed in manuscript by W.E. Sanford;
36. On page 411, in manuscript "Notes on gout cases" on 1 leaf of The Johns Hopkins Hospital letterhead form (Form 62. 10,000-12-29-1900);
37. On page 422, in manuscript a succinct reminder note on a cutout;
38. On page 478, in manuscript 1 leaf with initials R.H.J., dated Boston, 21 March, 1901;
39. On page 569, a manuscript letter to Dr. Osler from Joseph ...[?], dated April, 15th, 1900;
40. On page 577, clipping entitled: Late dysenterie abscess of the liver;
41. On back pastedown, a clipping from the Journal of Osteopathy, page 577-580, entitled: The use of drugs, H.H. McIntyre, M.D. [Hugh Henry McIntyre].

B.O. 3546

Tipped in volume 2:

1. Between front endpaper and flyleaf, manuscript letter to Dr. Osler, from Christian Bäumler, dated Freiburg i. Br., Nov. 27th 1898;
2. On front flyleaf, typescript business card for Docteur J. Dejerine [Joseph Jules Dejerine], Professeur Agrégé à la Faculté de Médecine, Médecin de la Salpétrière, with manuscript annotation dated 28.11.98;
3. On page 757, manuscript letter from Dr. Clarence H. Vaught of Richmond, KY, dated July 23, 1900 (2 pages);
4. On page 759, typescript letter on 1 leaf of The Johns Hopkins Hospital letterhead form (59-4000-7-23—96) addressed to Professor Lamberton [William A. Lamberton] from Henry M. Hurd, M.D., dated Baltimore, Jan. 29th, 1897, with a manuscript annotation signed by Charles N. Bancker Camac;
5. On page 759, manuscript letter on 1 leaf of University of Pennsylvania letterhead form addressed to Dr. Camac from William A. Lamberton, Dean, dated Philadelphia, March 2nd, 1897;
6. Opposite page 761, note in manuscript on a cutout;
7. On page 766, reprint from the October number of Colorado Medical Journal, entitled: A case of persistent foramen ovale with auscultatory signs by Henry Sewall (2 pages);
8. On page 769, typescript letter addressed to Dr. Osler, from Dr. George Dock, Ann Arbor, Mich., with manuscript signature, dated Jan. 19, 1901;
9. On page 779, clipping from Northwestern Lancet, pages 221-224, article entitled: Diagnosis of thoracic aneurism by Andrew Henderson, M.D, with manuscript note to W.O. from M.A.;
10. On page 788, 3 leaves of notes on The Johns Hopkins Hospital letterhead form (Form 72. 5000-5-15-1901);
11. On page 789, 1 leaf in typescript (with manuscript annotations) entitled: Congenital aneurism or periarteritis nodosa;
12. On page 837, manuscript note on cutout signed by Walter R. Steiner;
13. On page 1040, typescript letter addressed to Doctor Osler from War Department, Surgeon General's Office, U.S. Army Medical Museum and Library, dated Washington, D.C., December 18, 1900 (189) with manuscript signature by Robert Fletcher;
14. On page 1069, post-card in manuscript from Aurora, dated July 12, 1900, signed C. H. Maxwell , M.D. [Cyrus H. Maxwell];
15. On page 1110, pamphlet in printed paper wrappers, entitled: Tetany in infancy: with a report of six cases by John Lovett Morse, reprinted from The Journal of the American Medical Association, November 5, 1898 (16 pages);
16. On page 1139 laid in, clipping from Medical Review, dated January 6, 1900 entitled: Erythromelalgy by Rosin;
17. On page 1147, manuscript note, on form letter, with typescript caption, 1412 Eutaw Place, addressed to Dr. Osler, and signed Miss P. Hamhuger[?].

tion of an ulcer of the stomach. In Fitz's monograph 15 cases are reported. Körte has increased this number to 40. Symptoms of hæmorrhagic pancreatitis may precede or be associated with it. Death usually follows in from ten to twenty days, with symptoms of collapse.

Anatomically the pancreas may present a dry necrotic appearance, but as a rule the organ is converted into a dark slaty-colored mass lying nearly free in the omental cavity or attached by a few shreds. In other instances the totally or partially sequestrated organ may lie in a large abscess cavity, forming a palpable tumor in the epigastric region. In two cases, reported by Chiari, the necrotic pancreas was discharged per rectum, with recovery.

Relation of Fat-necrosis to Pancreatic Disease.—In connection with all forms of pancreatic disease small yellowish areas, to which Balser first directed attention, may be found in the interlobular pancreatic tissue, in the mesentery, in the omentum, and in the abdominal fatty tissue generally. In slight grades they may be present without other changes, and they have been seen in the living without any disease of the gland being discovered. They are most frequently in the hæmorrhagic and necrotic forms of pancreatitis, less common in the suppurative. In the pancreas the lobules are seen to be separated by a dead-white necrotic tissue, which gives a remarkable appearance to the section. In the abdominal fat the areas are usually not larger than a pin's head; they at once attract attention, and may be mistaken, on superficial examination, for miliary tubercles or neoplasms. They may be larger; instances have been reported in which they were the size of a hen's egg. On section they have a soft, tallowy consistence. Langerhans has shown that this substance is a combination of lime with certain fatty acids. They may be crusted with lime, and in a man, aged eighty, who died of Bright's disease, I found the lobules of the pancreas entirely isolated by areas of fatty necrosis with extensive deposition of lime salts. There is no necessary etiological relation between disease of the pancreas and disseminated fatty necroses of the abdomen at the time the latter are discovered. Cases have been found accidentally in laparotomy for ovarian tumor and in instances in which the pancreas has been normal. They may be present in thin persons or in association with gall-stones. The *bacterium coli commune* was present in two instances, with diphtheritic colitis, examined by Welch, though in most cases the areas of necrosis are sterile. Langerhans produced fat-necrosis by injecting extract of pancreas into the peri-renal fatty tissue of a dog; and Hildebrand and Williams have shown experimentally that the fat-necroses are caused by certain constituents of the pancreatic juice, but not by trypsin. Flexner has demonstrated by chemical tests the existence of the fat-splitting ferment in peritoneal fat-necroses in recent human and experimental cases. The ferment (steapsin) disappears after five or six days in experimental necroses, and cannot be demonstrated in the lime-incrusted human ones. H. U. Williams has produced similar lesions in the subcutaneous fat by inserting bits of sterile pancreas beneath the skin. In their experimental studies Hildebrand, Williams, and Flexner, while they were able to produce fat-necroses by tying the veins of and sometimes lacerating the pancreas, never actually succeeded in reproducing the picture of hæmorrhagic and necrotic pancreatitis. This has recently been

accomplished by Hlava and Flexner by injecting artificial gastric juice and dilute solutions of hydrochloric acid into the duct of Wirsung. The very acutely developing cases in dogs may result fatally within twenty-four hours. The fat-necroses in these cases are caused not by the acids but by the fat-splitting ferment (Flexner).

It is well for surgeons to remember that in two cases at least the most serious symptoms of acute pancreatic disease have been found in association with only widespread fat-necrosis of the gland. In a case reported by Stockton and Williams a man, on his return journey from Europe, was seized with vomiting and pain, without fever, but with a very small pulse. The patient died soon after his arrival in America. The post mortem showed a pancreas 18 cm. long, at first sight normal, but showing on section most extensive fatty infiltration with fat-necrosis.

III. CHRONIC PANCREATITIS.

Dieckhoff recognizes two forms: (1) The most common, a chronic inflammation which extends from the ducts, and is met with in association with chronic catarrhal processes in the stomach and duodenum and in the bile-passages; (2) a chronic pancreatitis of hæmatogenous origin, resulting from toxic materials in the blood, particularly from alcohol and lues. The organ may be reduced in size and very hard, as in the atrophic sclerosis seen not infrequently in diabetes. Occasionally it is larger than normal, and may form a tumor readily palpable in the upper part of the abdomen. In connection with the diabetic form there may be pigmentary changes in association with a similar condition in the liver. The sclerosis may follow pancreatic calculi, and occasionally interstitial lipomatosis causes great wasting of the tissue of the gland.

The interest in atrophy of the pancreas relates *first* to the association with it of diabetes, which has been already considered; and *secondly* to the possibility of a chronic interstitial pancreatitis, particularly at the head of the organ, blocking the terminal part of the common bile-duct. Riedel refers to severe cases in which he found during operation for gall-stones the head of the pancreas enlarged and hard as stone, so that he dreaded the possibility of new growth; but two of his patients recovered and were well for years, and in the third the post mortem showed that the condition was one of chronic pancreatitis. In one of Körte's cases a small nodule of the gland involved in a chronic pancreatitis had pressed directly upon the ductus communis choledochus and caused the jaundice.

IV. PANCREATIC CYSTS.

Of 121 cases operated upon by surgeons 60 were in males and 56 in females; in 5 the sex was not given (Körte). Sixty-six of the cases occurred in the fourth decade. T. C. Railton's case (which is not in Körte's series), an infant aged six months, and Shattuck's case in a child of thir-

teen and a half months, are the youngest in the literature. According to the origin Körte recognizes three varieties.

(1) **Traumatic Cases.**—In this list of 33 cases 30 were in men and only 3 in women. Blows on the abdomen or constantly repeated pressure are the most common forms of trauma. One case followed severe massage. Usually with the onset there are inflammatory symptoms, pain, and vomiting, sometimes suggestive of peritonitis. The contents of the cyst are usually bloody, though in 13 of the traumatic cases it was clear or yellowish.

(2) **Cysts following Inflammatory Conditions.**—In 51 cases the trouble began gradually after attacks of dyspepsia with colic, simulating somewhat that of gall-stones. Occasionally the attack set in with very severe symptoms, suggestive of obstruction of the bowel. In this group the tumor appeared in 19 cases soon after the onset of the pain; in others it was delayed for a period of from a few weeks to two or three years. McPhedran has reported a remarkable instance in which the tumor developed in the epigastrium with signs of severe inflammation. It was opened and drained and believed to be a hydrops of the lesser peritoneal cavity. Three months later a second cyst developed, which appeared to spring directly from the pancreas.

(3) **Cysts without any Inflammatory or Traumatic Etiology.**—Of 33 cases in this group 26 were in women. A remarkable feature is the prolonged period of their existence—in one case for forty-seven years, in one for between sixteen and twenty years, in others for sixteen, nine, and eight years, in the majority for from two to four years.

Anatomically Körte recognizes (1) *retention cysts* due to plugging of the main duct; (2) *proliferation cysts* of the pancreatic tissue—the cystoadenoma; (3) *retention cysts* arising from the alveoli of the gland and of the smaller ducts, which become cut off and dilate in consequence of chronic interstitial pancreatitis; (4) *pseudo-cysts* following inflammatory or traumatic affections of the pancreas, usually the result of injury, causing hæmorrhage and hydrops of the lesser peritonæum.

Situation.—In its growth the cyst may (1) develop in the lesser peritonæum, push the stomach upward, and reach the abdominal wall between the stomach and the transverse colon; (2) more rarely the cyst appears above the lesser curvature and pushes the stomach downward; in both of these cases the situation of the tumor is high in the abdomen, but in (3) it may develop between the leaves of the transverse meso-colon and lie below both the colon and the stomach. The relation of these two organs to the tumor is variable, but in the majority of cases the stomach lies above and the transverse colon below the cyst. Occasionally, too, as in T. C. Railton's case, the cyst may develop from the tail of the pancreas and project far over in the left hypochondrium in the position of the spleen or of a renal tumor.

General Symptoms.—Apart from the features of onset already referred to, the patient may complain of no trouble whatever, particularly in the very chronic cases, unless the cyst reaches a very large size. Painful colicky attacks, with nausea and vomiting and progressive enlargement of the abdomen, have frequently been noted. Fatty diarrhœa from disturb-

ance of the function of the pancreas is rare. Sugar in the urine has been present in a number of cases. Increased secretion of the saliva, the so-called pancreatic salivation, is also rare. Pressure of the cyst may sometimes cause jaundice, and in rare instances dyspnœa. Very marked loss of flesh has been present in a number of cases. A remarkable feature often noticed has been the transitory disappearance of the cyst. In one of Halsted's cases the girth of the abdomen decreased from 43 to 31 inches in ten days with profuse diarrhœa. Sometimes the disappearance has followed blows.

Diagnosis.—The cyst occupies the upper abdomen, usually forming a semicircular bulging in the median line, rarely to either side. In 16 cases Körte states that the chief projection was below the navel. In one case operated upon by Halsted the tumor occupied the greater part of the abdomen. The cyst is immobile, respiration having little or no influence on it. As already mentioned, the stomach, as a rule, lies above it and the colon below.

In a majority of the cases the fluid is of a reddish or dark-brown color, and contains blood or blood coloring matter, cell detritus, fat granules, and sometimes cholesterin. The consistence of the fluid is usually mucoid, rarely thin. The reaction is alkaline, the specific gravity from 1.010 to 1.020. In 22 cases Körte states that the fluid was not hæmorrhagic.

The existence of ferments is important. In 54 cases they were present in the fluid or in the material from the fistula. In 20 cases only one ferment was present, in 20 cases two, and in 14 cases all three of the pancreatic ferments were found. As diastatic and fat emulsifying ferments occur widely in various exudates the most important and only positive signs in the diagnosis of the pancreatic secretion is the digestion of fibrin and albumin.

Results.—Körte states of 101 cases in which the cyst was opened and drained 4 deaths followed the operation directly; 1 resulted from infection of the fistula. In 14 cases the cyst was extirpated; of these 12 recovered.

V. TUMORS OF THE PANCREAS.

Of new growths in the organ carcinoma is the most frequent. Sarcoma, adenoma, and lymphoma are rare.

Frequency.—At the General Hospital in Vienna in 18,069 autopsies there were 22 cases of cancer of the pancreas (Biach). In 11,472 postmortems at Milan, Segré found 132 tumors of the pancreas, 127 of which were carcinomata, 2 sarcomata, 2 cysts, and 1 syphiloma. In 6,000 autopsies at Guy's Hospital there were only 20 cases of primary malignant disease of the organ (Hale White). In the first 1,000 autopsies at the Johns Hopkins Hospital there were 5 cases of adeno-carcinoma, and 1 doubtful case in which the exact origin could not be stated. There were 5 cases of secondary malignant disease of the pancreas. The head of the gland is most commonly involved, but the disease may be limited to the body or to the tail. The majority of the patients are in the middle period of life.

Symptoms.—The diagnosis is not often possible. The following are the most important and suggestive features: (*a*) Epigastric pains, often

occurring in paroxysms. (*b*) Jaundice, due to pressure of the tumor in the head of the pancreas on the bile-duct. The jaundice is intense and permanent, and associated with dilatation of the gall-bladder, which may reach a very large size. (*c*) The presence of a tumor in the epigastrium. This is very variable. In 137 cases Da Costa found the tumor present in only 13. Palpation under anæsthesia with the stomach empty would probably give a very much larger percentage. As the tumor rests directly upon the aorta there is usually a marked degree of pulsation, sometimes with a bruit. There may be pressure on the portal vein, causing thrombosis and its usual sequels. (*d*) Symptoms due to loss of function of the pancreas are less important. Fatty diarrhœa is not very often present. In consequence of the absence of bile the stools are usually very clay-colored and greasy. Diabetes also is not common. (*e*) A very rapid wasting and cachexia. Of other symptoms nausea and vomiting are common. In some instances the pylorus is compressed and there is great dilatation of the stomach. In a few cases there has been profuse salivation.

The points of greatest importance in the diagnosis are the intense and permanent jaundice, with dilatation of the gall-bladder, rapid emaciation, and the presence of a tumor in the epigastric region. Of less importance are features pointing to disturbance of the function of the gland.

Of other new growths sarcoma and lymphoma have been occasionally found. Miliary tubercle is not very uncommon in the gland. Syphilis may occur as rather a chronic interstitial inflammation, or in the form of gummous tumors.

The outlook in tumors of the pancreas is, as a rule, hopeless. However, of 10 cases operated upon of late years, 6 recovered (Körte).

VI. PANCREATIC CALCULI.

Pancreatic lithiasis is comparatively rare. In 1883 George W. Johnston collected 35 cases in the literature. In 1,000 autopsies at the Johns Hopkins Hospital there were 2 cases.

The stones are usually numerous, either round in shape or rough, spinous and coral-like. The color is opaque white. They are composed chiefly of carbonate of lime. The effects of the stones are: (1) A chronic interstitial inflammation of the gland substance with dilatation of the duct; sometimes there is cystic dilatation of the gland; (2) acute inflammation with suppuration; (3) the irritation of the stones, as in the gall-bladder, may lead to carcinoma.

Symptoms.—Pepper in 1882 made a diagnosis of calculus of the pancreas, of which, however, there was no confirmation either by the passage of the stone or by autopsy. Minnich has reported a case in which, after an attack of colic, calculi composed of calcic carbonate and phosphate were passed in the stools. Lichtheim, in a case with severe colic, diabetes, and fatty diarrhœa, made the diagnosis of pancreatic calculi, which was afterward confirmed by autopsy.

X. DISEASES OF THE PERITONÆUM.

I. ACUTE GENERAL PERITONITIS.

Definition.—Acute inflammation of the peritonæum.

Etiology.—The condition may be primary or secondary.

(*a*) **Primary, Idiopathic Peritonitis.**—Considering how frequently the pleura and pericardium are primarily inflamed the rarity of idiopathic inflammation of the peritonæum is somewhat remarkable. It may follow cold or exposure and is then known as rheumatic peritonitis. No instance of the kind has come under my notice. In Bright's disease, gout, and arterio-sclerosis acute peritonitis may develop as a terminal event. Of 102 cases of peritonitis which came to autopsy at the Johns Hopkins Hospital, 12 were of this form. In these there was some pre-existing chronic disease (Flexner).

(*b*) **Secondary peritonitis** is due to extension of inflammation from, or perforation of one of the organs covered by the peritonæum. Peritonitis from extension may follow inflammation of the stomach or intestines, extensive ulceration in these parts, cancer, acute suppurative inflammations of the spleen, liver, pancreas, retroperitoneal tissues, and the pelvic viscera.

Perforative peritonitis is the most common, following external wounds, perforation of ulcer of the stomach or bowels, perforation of the gall-bladder, abscess of the liver, spleen, or kidneys. Two important causes are appendicitis and suppurating inflammation about the Fallopian tubes and ovaries. There are instances in which peritonitis has followed rupture of an apparently normal Graafian follicle.

Of the above 102 cases, 56 originated in an extension from some diseased abdominal viscus. The remaining 34 followed surgical operations upon the peritonæum or the contained organs.

The peritonitis of septicæmia and pyæmia is almost invariably the result of a local process. An exceedingly acute form of peritonitis may be caused by the development of tubercles on the membrane.

Morbid Anatomy.—In recent cases, on opening the abdomen the intestinal coils are distended and glued together by lymph, and the peritonæum presents a patchy, sometimes a uniform injection. The exudation may be: (*a*) Fibrinous, with little or no fluid, except a few pockets of clear serum between the coils. (*b*) Sero-fibrinous. The coils are covered with lymph, and there is in addition a large amount of a yellowish, sero-fibrinous fluid. In instances in which the stomach or intestine is perforated this may be mixed with food or fæces. (*c*) Purulent, in which the exudate is either thin and greenish yellow in color, or opaque white and creamy. (*d*) Putrid. Occasionally in puerperal and perforative peritonitis, particularly when the latter has been caused by cancer, the exudate is thin, grayish green in color, and has a gangrenous odor. (*e*) Hæmorrhagic. This is sometimes found as an admixture in cases of acute peritonitis following wounds, and occurs in the cancerous and tuberculous

forms. (*f*) A rare form occurs in which the injection is present, but almost all signs of exudation are wanting. Close inspection may be necessary to detect a slight dulling of the serous surfaces. The bacteriological examination reveals large numbers of bacteria.

The amount of the effusion varies from half a litre to 20 or 30 litres. There are probably essential differences between the various kinds of peritonitis.

Bacteriology of Acute Peritonitis.—Much work has been done lately upon the subject. Flexner has analyzed 102 cases of peritonitis, in which bacteriological studies were made, which came to autopsy in the Johns Hopkins Hospital. He makes three classes. The first class embraces the primary or idiopathic form, of which 12 cases were found. These were with one exception mono-infections. The prevailing micro-organism was the streptococcus pyogenes (five times), the remaining ones being the staphylococcus aureus, micrococcus lanceolatus, bacillus proteus, pyocyaneus, and coli communis. The second class followed operations upon the peritonæum, excepting operations upon the intestine. The majority of these cases were examples of wound infection. They were 33 in number. In 25 of these mono-infections, in 8 mixed infections existed. The prevailing micro-organism was the staphylococcus aureus, which was present alone in 12 and combined in 2 cases. The streptococcus occurred 5 times uncombined and 4 times combined. The bacillus coli was found 5 times in all, being unassociated in 3 cases. Other organisms found were the micrococcus lanceolatus, staphylococcus albus, bacillus pyocyaneus, and ærogenes capsulatus. The remaining 56 cases, forming the third class, were instances of intestinal infection. These comprised 23 mono- and 33 polyinfections. The predominating micro-organism was the bacillus coli communis which occurred in 43 cases, 8 times alone and 35 in association. The streptococcus was present in 37 cases, being alone in 7. The staphylococci, pneumococcus, bacillus proteus, pyocyaneus, typhosus, and aerogenes capsulatus occurred in a smaller number of instances.

Among the micro-organisms thus far found rarely in peritonitis, may be mentioned the gonococcus, the anthrax bacillus, the proteus bacillus, and the typhoid bacillus. As illustrating the importance of the gonococcus, I may state that as I write there are two young girls both of whom were admitted to my wards with diffuse peritonitis arising from fresh gonorrhœal salpingitis. Both were operated upon by Cushing successfully. Welch has found the bacillus coli communis in peritonitis due to ulceration of the intestines without perforation.

Symptoms.—In the perforative and septic cases the onset is marked by chilly feelings or an actual rigor with intense pain in the abdomen. In typhoid fever, when the sensorium is benumbed, the onset may not be noticed. The pain is general, and is usually intense and aggravated by movements and pressure. A position is taken which relieves the tension of the abdominal muscles, so that the patient lies on the back with the thighs drawn up and the shoulders elevated. The greatest pain is usually below the umbilicus, but in peritonitis from perforation of the stomach pain may be referred to the back, the chest, or the shoulder. The respira-

tion is superficial—costal in type—as it is painful to use the diaphragm. For the same reason the action of coughing is restrained, and even the movements necessary for talking are limited. In this early stage the sensitiveness may be great and the abdominal muscles are often rigidly contracted. If the patient is at perfect rest the pain may be very slight, and there are instances in which it is not at all marked, and may, indeed, be absent.

The abdomen gradually becomes distended and tense and is tympanitic on percussion. The pulse is rapid, small, and hard, and often has a peculiar wiry quality. It ranges from 110 to 150. The temperature may rise rapidly after the chill and reach 104° or 105°, but the subsequent elevation is moderate. In some very severe cases there may be no fever throughout. The tongue at first is white and moist, but subsequently becomes dry and often red and fissured. Vomiting is an early and prominent feature and causes great pain. The contents of the stomach are first ejected, then a yellowish and bile-stained fluid, and finally a greenish and, in rare instances, a brownish-black liquid with slight fæcal odor. The bowels may be loose at the onset and then constipation may follow. Frequent micturition may be present, less often retention. The urine is usually scanty and high-colored, and contains a large quantity of indican.

The appearance of the patient when these symptoms have fully developed is very characteristic. The face is pinched, the eyes are sunken, and the expression is very anxious. The constant vomiting of fluids causes a wasted appearance, and the hands sometimes present the washer-woman's skin. Except in cholera, we see the Hippocratic facies more frequently in this than in any other disease—"*a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the color of the whole face being brown, black, livid, or lead-colored.*" There are one or two additional points about the abdomen. The tympany is usually excessive, owing to the great relaxation of the walls of the intestines by inflammation and exudation. The splenic dulness may be obliterated, the diaphragm pushed up, and the apex beat of the heart dislocated to the fourth interspace. The liver dulness may be greatly reduced, or may, in the mammary line, be obliterated. It has been claimed that this is a distinctive feature of perforative peritonitis, but on several occasions I have been able to demonstrate that the liver dulness in the middle and mammary line was obliterated by tympanites alone. In the axillary line, on the other hand, the liver dulness, though diminished, may persist. Pneumo-peritonæum following perforation more certainly obliterates the hepatic dulness. In such cases the fluid effused produces a dulness in the lateral region; but with gas in the peritonæum, if the patient is turned on the left side, a clear note is heard beneath the seventh and eighth ribs. Acute peritonitis may present a flat, rigid abdomen throughout its course.

Effusion of fluid—ascites—is usually present except in some acute rapidly fatal cases. The flanks are dull on percussion. The dulness may be movable, though this depends altogether upon the degree of adhesions. There may be considerable effusion without either movable dulness or

fluctuation. A friction-rub may be present, as first pointed out by Bright, but it is not nearly so common in acute as in chronic peritonitis.

Course.—The acute diffuse peritonitis usually terminates in death. The most intense forms may kill within thirty-six to forty-eight hours; more commonly death results in four or five days, or the attack may be prolonged to eight or ten days. The pulse becomes irregular, the heart-sounds weak, the breathing shallow; there are lividity with pallor, a cold skin with high rectal temperature—a group of symptoms indicating profound failure of the vital functions for which Gee has revived the old term *lipothymia*. Occasionally death occurs with great suddenness, owing, possibly, to paralysis of the heart.

Diagnosis.—In typical cases the severe pain at onset, the distention of the abdomen, the tenderness, the fever, the gradual development of effusion, collapse symptoms, and the vomiting give a characteristic picture. Careful inquiries should at once be made concerning the previous condition, from which a clew can often be had as to the starting-point of the trouble. In young adults a considerable proportion of all cases depends upon perforating appendicitis, and there may be an account of previous attacks of pain in the iliac region, or of constipation alternating with diarrhœa. In women the most frequent causes are suppurative processes in the pelvic viscera, associated with salpingitis, abscesses in the broad ligaments, or acute puerperal infection. Perforation of gastric ulcer is a more common factor in women than in men. It is not always easy to determine the cause. Many cases come under observation for the first time with the abdomen distended and tender, and it is impossible to make a satisfactory examination. In such instances the pelvic organs should be examined with the greatest care. In typhoid fever, if the patient is conscious, the sudden onset of pain, the development of great meteorism, and the aggravation of the general symptoms indicate clearly what has happened. When the patient is in deep coma, on the other hand, the perforation may be overlooked. The following conditions are most apt to be mistaken for acute peritonitis:

(*a*) *Acute Entero-colitis.*—Here the pain and distention and the sensitiveness on pressure may be marked. The pain is more colicky in character, the diarrhœa is more frequent, and the collapse is more extreme.

(*b*) *The So-called Hysterical Peritonitis.*—This has deceived the very elect, as almost every feature of genuine peritonitis, even the collapse, may be simulated. The onset may be sudden, with severe pain in the abdomen, tenderness, vomiting, diarrhœa, difficulty in micturition, and the characteristic decubitus. Even the temperature may be elevated. There may be recurrence of the attack. A case has been reported by Bristowe in which four attacks occurred within a year, and it was not until special hysterical symptoms developed that the true nature of the trouble was suspected.

(*c*) *Obstruction of the bowel*, as already mentioned, may simulate peritonitis, both having pain, vomiting, tympanites, and constipation in common. It may for a couple of days really be impossible to make a diagnosis in the absence of a satisfactory history.

(*d*) *Rupture of an abdominal aneurism or embolism of the superior*

mesenteric artery may cause symptoms which simulate peritonitis. In the latter, sudden onset with severe pain, the collapse symptoms, frequent vomiting, and great distention of the abdomen may be present.

(*e*) I have already referred to the fact that acute hæmorrhagic pancreatitis may be mistaken for peritonitis. Lastly, a ruptured tubal pregnancy may resemble acute peritonitis.

II. PERITONITIS IN INFANTS.

Peritonitis may occur in the fœtus as a consequence of syphilis, and may lead to constriction of the bowel by fibrous adhesions.

In the new-born a septic peritonitis may extend from an inflamed cord. Distention of the abdomen, slight swelling and redness about the cord, and not infrequently jaundice are present. It is an uncommon event, and existed in only 4 of 51 infants dying with inflammation of the cord and septicæmia (Runge).

During childhood peritonitis develops from causes similar to those affecting the adult. Perforative appendicitis is common. Peritonitis following blows or kicks on the abdomen occurs more frequently at this period. In boys injury while playing foot-ball may be followed by diffuse peritonitis. A rare cause in children is extension through the diaphragm from an empyema. There are on record instances of peritonitis occurring in several children at the same school, and it has been attributed to sewer-gas poisoning. It was in investigating an epidemic of this kind at the Wandsworth school, in London, that Anstie received the post-mortem wound of which he died.

III. LOCALIZED PERITONITIS.

1. Subphrenic Peritonitis.—The general peritonæum covering the right and left lobes of the liver may be involved in an extension from the pleura of suppurative, tuberculous, or cancerous processes. In various affections of the liver—cancer, abscess, hydatid disease, and in affections of the gall-bladder—the inflammation may be localized to the peritonæum covering the upper surface of the organ. These forms of localized subphrenic peritonitis in the greater sac are not so important in reality as those which occur in the lesser peritonæum. The anatomical relations of this structure are as follows: It lies behind and below the stomach, the gastro-hepatic omentum, and the anterior layer of the great omentum. Its lower limit forms the upper layer of the transverse meso-colon. On either side it reaches from the hepatic to the splenic flexure of the colon, and from the foramen of Winslow to the hilus of the spleen. Behind it covers and is tightly adherent to the front of the pancreas. Its upper limit is formed by the transverse fissure of the liver, and by that portion of the diaphragm which is covered by the lower layer of the right lateral ligament of the liver; the lobus Spigelii lies bare in the cavity. The foramen

of Winslow, through which the lesser communicates with the greater peritonæum, is readily closed by inflammation.

Inflammatory processes, exudates, and hæmorrhages may be confined entirely to the lesser peritonæum. The exudate of tuberculous peritonitis may be confined to it. Perforations of certain parts of the stomach, of the duodenum, and of the colon may excite inflammation in it alone; and in various affections of the pancreas, particularly trauma and hæmorrhage, the effusion into the sac has often been confounded with cyst of this organ. "Pathological distention of the lesser peritonæum gives rise to a tumor in the left hypochondriac, epigastric, and umbilical regions of a somewhat characteristic shape, but which appears to vary from time to time in form and size, according to the conditions of the overlying stomach; for when the viscus is full of liquid contents it increases the area of the tumor's dulness, while it makes its outlines less definable by palpation, and if the stomach is distended with gas the dull area becomes resonant and apparently the tumor may disappear altogether. The colon always lies below the tumor and never in front of or above it, as is the case in kidney enlargement" (Jordan Lloyd).

Special mention must be made of the remarkable form of subphrenic abscess containing air, which may simulate closely pneumothorax, and hence was called by Leyden *Pyo-pneumothorax subphrenicus*. The affection has been thoroughly studied of late years by Scheurlen, Mason, Meltzer, and Lee Dickinson. In 142 out of 170 recorded cases the cause was known. In a few instances, as in one reported by Meltzer, the subphrenic abscess seemed to have followed pneumonia. Pyothorax is an occasional cause. By far the most frequent condition is gastric ulcer, which occurred in 80 of the cases. Duodenal ulcer was the cause in 6 per cent. In about 10 per cent of the cases the appendix was the starting-point of the abscess. Cancer of the stomach is an occasional cause. Other rare causes are trauma, which was present in one of my cases, perforation of an hepatic or a renal abscess, lesions of the spleen, abscess, and cysts of the pancreas.

In a majority of all the cases in which the stomach or duodenum is perforated—sometimes, indeed, in the cases following trauma, as in *Case 3* of my series—the abscess contains air.

The symptoms of subphrenic abscess vary very considerably, depending a good deal upon the primary cause. The onset, as a rule, is abrupt, particularly when due to perforation of a gastric ulcer. There are severe pain, vomiting, often of bilious or of bloody material; respiration is embarrassed, owing to the involvement of the diaphragm; then the constitutional symptoms develop associated with suppuration, chills, irregular fever, and emaciation. Subsequently perforation may take place into the pleura or into the lung, with severe cough and abundant purulent expectoration.

The conditions are so obscure that the diagnosis of subphrenic abscess is not often made. The perihepatic abscess beneath the arch of the diaphragm, whether to the right or left of the suspensory ligament, when it does not contain air, is almost invariably mistaken for empyema. When a pus collection of any size is in the lesser peritonæum, the tumor is formed

which has the characters already mentioned in a quotation from Mr. Jordan Lloyd.

The most remarkable features are those which are superadded when the abscess cavity contains air. Here, on the right side, when the abscess is in the greater peritonæum, above the right lobe of the liver, the diaphragm may be pushed up to the level of the second or third rib, and the physical signs on percussion and auscultation are those of pneumothorax, particularly the tympanitic resonance and the movable dulness. The liver is usually greatly depressed and there is bulging on the right side. Still more obscure are the cases of air-containing abscesses due to perforation of the stomach or duodenum, in which the gas is contained in the lesser peritonæum. Here the diaphragm is pushed up and there are signs of pneumothorax on the left side. In a large majority of all the cases which follow perforation of a gastric ulcer the effusion lies between the diaphragm above, and the spleen, stomach, and the left lobe of the liver below.

The prognosis in subphrenic abscess is not very hopeful. Of the cases on record about 20 per cent only have recovered. Of the five cases which have come under my observation, three recovered after operation.

2. **Appendicular.**—The most frequent cause in the male of localized peritonitis is inflammation of the appendix vermiformis. The situation varies with the position of this extremely variable organ. The adhesion, perforation, and intraperitoneal abscess cavity may be within the pelvis, or to the left of the median line in the iliac region, in the lower right quadrant of the umbilical region—a not uncommon situation—or, of course, most frequently in the right iliac fossa. In the most common situation the localized abscess lies upon the psoas muscle, bounded by the cæcum on the right and the terminal portion of the ileum and its mesentery in front and to the left. In many of these cases the limitation is perfect, and post-mortem records show that complete healing may take place with the obliteration of the appendix in a mass of firm scar tissue.

3. **Pelvic Peritonitis.**—The most frequent cause is inflammation about the uterus and Fallopian tubes. Puerperal septicæmia, gonorrhœa, and tuberculosis are the usual causes. The tubes are the starting-point in a majority of the cases. The fimbriæ become adherent and closely matted to the ovary, and there is gradually produced a condition of thickening of the parts, in which the individual organs are scarcely recognizable. The tubes are dilated and filled with cheesy matter or pus, and there may be small abscess cavities in the broad ligaments. Rupture of one of these may cause general peritonitis, or the membrane may be involved by extension, as in tuberculosis of these parts.

IV. CHRONIC PERITONITIS.

The following varieties may be recognized: (*a*) **Local adhesive peritonitis,** a very common condition, which occurs particularly about the spleen, forming adhesions between the capsule and the diaphragm, about the liver,

less frequently about the intestines and mesentery. Points of thickening or puckering on the peritonæum occur sometimes with union of the coils or with fibrous bands. In a majority of such cases the condition is met accidentally post mortem. Two sets of symptoms may, however, be caused by these adhesions. When a fibrous band is attached in such a way as to form a loop or snare, a coil of intestine may pass through it. Thus, of the 295 cases of intestinal obstruction analyzed by Fitz, 63 were due to this cause. The second group is less serious and comprises cases with persistent abdominal pain of a colicky character, sometimes rendering life miserable. Instances of this kind have been successfully operated upon by Homans and H. A. Kelly.

(*b*) **Diffuse Adhesive Peritonitis.**—This is a consequence of an acute inflammation, either simple or tuberculous. The peritonæum is obliterated. On cutting through the abdominal wall, the coils of intestines are uniformly matted together and can neither be separated from each other nor can the visceral and parietal layers be distinguished. There may be thickening of the layers, and the liver and spleen are usually involved in the adhesions.

(*c*) **Proliferative Peritonitis.**—Apart from cancer and tubercle, which produce typical lesions of chronic peritonitis, the most characteristic form is that which may be described under this heading. The essential anatomical feature is great thickening of the peritoneal layers, usually without much adhesion. The cases are sometimes seen with sclerosis of the stomach. In one instance I found it in connection with a sclerotic condition of the cæcum and the first part of the colon. In the inspection of a case of this kind there is usually moderate effusion, more rarely extensive ascites. The peritonæum is opaque-white in color, and everywhere thickened, often in patches. The omentum is usually rolled and forms a thickened mass transversely placed between the stomach and the colon. The peritonæum over the stomach, intestines, and mesentery is sometimes greatly thickened. The liver and spleen may simply be adherent, or there is a condition of chronic perihepatitis or perisplenitis, so that a layer of firm, almost gristly connective tissue of from one fourth to half an inch in thickness encircles these organs. Usually the volume of the liver is in consequence greatly reduced. The gastro-hepatic omentum may be constricted by this new growth and the calibre of the portal vein much narrowed. A serous effusion may be present. On account of the adhesions which form, the peritonæum may be divided into three or four different sacs, as is more fully described under the tuberculous peritonitis. In these cases the intestines are usually free, though the mesentery is greatly shortened. There are instances of chronic peritonitis in which the mesentery is so shortened by this proliferative change that the intestines form a ball not larger than a cocoa-nut situated in the middle line, and after the removal of the exudation can be felt as a solid tumor. The intestinal wall is greatly thickened and the mucous membrane of the ileum is thrown into folds like the valvulæ conniventes. This proliferative peritonitis is found frequently in the subjects of chronic alcoholism. In cases of long-continued ascites the serous surfaces generally become thickened and present an opaque, dead-white

color. This condition is observed especially in hepatic cirrhosis, but attends tumors, chronic passive congestions, etc.

In all forms of chronic peritonitis a friction may be felt usually in the upper zone of the abdomen.

In some instances of chronic peritonitis the membrane presents numerous nodular thickenings, which may be mistaken for tubercles. They may be scattered in numbers on the membranes, and it may be extremely difficult, without the most careful microscopical examination, to determine their nature. J. F. Payne has described a case of this sort associated with disseminating growths throughout the liver which were not cancerous. It has been suggested that some of the cases of tuberculous peritonitis cured by operation have been of this nature, but histological examination would, as a rule, readily determine between the conditions. Miura, in Japan, has reported a case in which these nodules contained the ova of a parasite. One case has been reported in which the exciting cause was regarded as cholesterin plates, which were contained within the granulomatous nodules.

(*d*) **Chronic Hæmorrhagic Peritonitis.**—Blood-stained effusions in the peritonæum occur particularly in cancerous and tuberculous disease. There is a form of chronic inflammation analogous to the hæmorrhagic pachymeningitis of the brain. It was described first by Virchow, and is localized most commonly in the pelvis. Layers of new connective tissue form on the surface of the peritonæum with large wide vessels from which hæmorrhage occurs. This is repeated from time to time with the formation of regular layers of hæmorrhagic effusion. It is rarely diffuse, more commonly circumscribed.

V. NEW GROWTHS IN THE PERITONÆUM.

(*a*) **Tuberculous Peritonitis.**—This has already been considered.

(*b*) **Cancer of the Peritonæum.**—Although, as a rule, secondary to disease of the stomach, liver, or pelvic organs, cases of primary cancer have been described. It is probable that the so-called primary cancers of the serous membranes are endotheliomata and not carcinomata. Secondary malignant peritonitis occurs in connection with all forms of cancer. It is usually characterized by a number of round tumors scattered over the entire peritonæum, sometimes small and miliary, at other times large and nodular, with puckered centres. The disease most commonly starts from the stomach or the ovaries. The omentum is indurated, and, as in tuberculous peritonitis, forms a mass which lies transversely across the upper portion of the abdomen. Primary malignant disease of the peritonæum is extremely rare. Colloid is said to have occurred, forming enormous masses, which in one case weighed over 100 pounds. Cancer of this membrane spreads, either by the detachment of small particles which are carried in the lymph currents and by the movements to distant parts, or by contact of opposing surfaces. It occurs more frequently in women than in men, and more commonly at the later period of life.

The *diagnosis* of cancer of the peritonæum is easy with a history of a

local malignant disease; as when it occurs with ovarian tumor or with cancer of the pylorus. In cases in which there is no evidence of a primary lesion the diagnosis may be doubtful. The clinical picture is usually that of chronic ascites with progressive emaciation. There may be no fever. If there is much effusion nothing definite can be felt on examination. After tapping, irregular nodules or the curled omentum may be felt lying transversely across the upper portion of the abdomen. Unfortunately, this tumor upon which so much stress is laid occurs as frequently in tuberculous peritonitis and may be present in a typical manner in the chronic proliferative form, so that in itself it has no special diagnostic value. Multiple nodules, if large, indicate cancer, particularly in persons above middle life. Nodular tuberculous peritonitis is most frequent in children. The presence about the navel of secondary nodules and indurated masses is more common in cancer. Inflammation, suppuration, and the discharge of pus from the navel rarely occurs except in tuberculous disease. Considerable enlargement of the inguinal glands may be present in cancer. The nature of the fluid in cancer and in tubercle may be much alike. It may be hæmorrhagic in both; more often in the latter. The histological examination in cancer may show large multinuclear cells or groups of cells—the sprouting cell-groups of Foulis—which are extremely suggestive. The colloid cancer may produce a totally different picture; instead of ascitic fluid, the abdomen is occupied by the semi-solid gelatinous substance, and is firm, not fluctuating.

And, lastly, there are instances of echinococci in the peritonæum which may simulate cancer very closely. I have reported a case of this kind, in which the enlarged liver and the innumerable nodular masses in the peritonæum naturally led to this diagnosis.

VI. ASCITES (*Hydro-peritonæum*).

Definition.—The accumulation of serous fluid in the peritoneal cavity.

Etiology.—(1) **Local Causes.**—(*a*) Chronic inflammation of the peritonæum, either simple, cancerous, or tuberculous. (*b*) Portal obstruction in the terminal branches within the liver, as in cirrhosis and chronic passive congestion, or by compression of the vein in the gastro-hepatic omentum, either by proliferative peritonitis, by new growths, or by aneurism. (*c*) Tumors of the abdomen. The solid growths of the ovaries may cause considerable ascites, which may completely mask the true condition. The enlarged spleen in leukæmia, less commonly in malaria, may be associated with recurring ascites.

(2) **General Causes.**—The ascites is part of a general dropsy, the result of mechanical effects, as in heart-disease, chronic emphysema, and sclerosis of the lung. In cardiac lesions the effusion is sometimes confined to the peritonæum, in which case it is due to secondary changes in the liver, or it has been suggested to be connected with a failure of the suction action of this organ, by which the peritonæum is kept dry. Ascites occurs also in the dropsy of Bright's disease, and in hydræmic states of the blood.

Symptoms.—A gradual uniform enlargement of the abdomen is the characteristic symptom of ascites. The physical signs are usually distinctive. (*a*) *Inspection.*—According to the amount of fluid the abdomen is protuberant and flattened at the sides. With large effusions, the skin is tense and may present the lineæ albicantes. Frequently the navel itself and the parts about it are very prominent. In many cases the superficial veins are enlarged and a plexus joining the mammary vessels can be seen. Sometimes it can be determined by pressure on these veins that the current is from below upward. In some instances, as in thrombosis or obliteration of the portal vein, these superficial abdominal vessels may be extensively varicose. About the navel in cases of cirrhosis there is occasionally a large bunch of distended veins, the so-called caput Medusæ.

(*b*) *Palpation.*—Fluctuation is obtained by placing the fingers of one hand upon one side of the abdomen and by giving a sharp tap on the opposite side with the other hand, when a wave is felt to strike as a definite shock against the applied fingers. Even comparatively small quantities of fluid may give this fluctuation shock. When the abdominal walls are thick or very fat, an assistant may place the edge of the hand or a piece of cardboard in the front of the abdomen. A different procedure is adopted in palpating for the solid organs in case of ascites. Instead of placing the hand flat upon the abdomen, as in the ordinary method, the pads of the fingers only are placed lightly upon the skin, and then by a sudden depression of the fingers the fluid is displaced and the solid organ or tumor may be felt. By this method of "dipping" or displacement, as it is called, the liver may be felt below the costal margin, or the spleen, or sometimes solid tumors of the omentum or intestine.

(*c*) *Percussion.*—In the dorsal position with a moderate quantity of fluid in the peritonæum the flanks are dull, while the umbilical and epigastric regions, into which the intestines float, are tympanitic. This area of clear resonance may have an oval outline. Having obtained the lateral limit of the dulness on one side, if the patient turns on the opposite side, the fluid gravitates to the dependent part and the uppermost flank is now tympanitic. In moderate effusions this movable dulness changes greatly in the different postures. Small amounts of fluid, probably under a litre, would scarcely give movable dulness, as the pelvis and the renal regions hold a considerable quantity. In such cases it is best to place the patient in the knee-elbow position, when a dull note will be determined at the most dependent portion. By careful attention to these details mistakes are usually avoided.

The following are among the conditions which may be mistaken for dropsy: *Ovarian tumor*, in which the sac develops, as a rule, unilaterally, though when large it is centrally placed. The dulness is anterior and the resonance is in the flanks, into which the intestines are pushed by the cyst. Examination *per vaginam* may give important indications. In those rare instances in which gas develops in the cyst the diagnosis may be very difficult. Succussion has been obtained in such cases. A *distended bladder* may reach above the umbilicus. In such instances some urine dribbles away, and suspicion of ascites or a cyst is occasionally entertained. I once

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saw a trochar thrust into a distended bladder, which was supposed to be an ovarian cyst, and it is stated that John Hunter tapped a bladder, supposing it to be ascites. Such a mistake should be avoided by careful catheterization prior to any operative procedures. And lastly, there are large pancreatic or hydatid cysts in the abdomen which may simulate ascites.

Nature of the Ascitic Fluid.—Usually this is a clear serum, light yellow in the ascites of anæmia and Bright's disease, often darker in color in cirrhosis of the liver. The specific gravity is low, seldom more than 1.010 or 1.015, whereas in the fluid of ovarian cysts the specific gravity is high, 1,020 or over. It is albuminous and sometimes coagulates spontaneously. Dock has called attention to the importance of the study of the cells in the exudate. In cancer very characteristic forms, with nuclear figures, may be found. Hæmorrhagic effusion usually occurs in cancer and tuberculosis, and occasionally in cirrhosis. I have already referred to the instances of hæmorrhagic effusion in connection with ruptured tubal pregnancy. A chylous, milky exudate is occasionally found. Busey has collected 33 cases from the literature. There are, as Quincke has pointed out, two distinct varieties, a fatty and a chylous, which may be distinguished by the microscope, as in the former there are distinct fat-globules. These cases have been sometimes connected with peritoneal or mesenteric cancer. In the true chylous ascites the fluid is turbid and milky. In some of the cases, as in Whitla's, a perforation of the thoracic duct has been found. The condition does not necessarily follow obliteration of the thoracic duct. Mild grades of chylous ascites, which are occasionally found clinically, may be due to the fact that the patient upon a milk diet has a permanent lipæmia, such as is present in young animals and in diabetics, in whom the liquor sanguinis is always fatty. Under such circumstances an exudate may contain enough of the molecular base of the chyle to produce turbidity of the fluid. Some of the cases have been associated with filariasis. In a recent case in my clinic N. McL. Harris isolated the bacillus diphtheriæ from the chylous fluid.

Treatment of the Previous Conditions.—(*a*) **Acute Peritonitis.**—Rest is enjoined upon the patient by the severe pain which follows the slightest movement, and he should be propped in the position which gives him greatest relief. For the pain morphia should be injected hypodermically in full doses. In an adult it is better to give a third or half a grain at once, and subsequently at intervals repeat it in smaller doses, when necessary. The action of the drug should be carefully watched and the patient should not be allowed to pass into such a degree of unconsciousness that he cannot be aroused. The respiration and the condition of the pupils also give valuable information. The amount of opium which has been given in certain instances is remarkable, and indicates a tolerance of the drug. The doses given by the late Alonzo Clark, of New York, may be truly termed heroic. Austin Flint notes that a patient under the care of this physician took "in the first twenty-four hours, of opium and the sulphate of morphia, a quantity equivalent to 106 grains of opium; in the second twenty-four hours she took 472 grains; on the third day, 236 grains; on the fourth day, 120 grains; on the fifth day,

54 grains; on the sixth day, 22 grains; on the seventh day, 18 grains; after which the treatment was suspended." It is unnecessary to use these enormous doses, as, even when the pain is most intense, from a third to a half grain of morphia every few hours will usually keep the patient thoroughly under the influence of the drug. In a robust, strong patient, seen at the outset, twenty leeches applied over the abdomen will give great relief.

Local applications—either hot turpentine stupes or cloths wrung out of ice-water—may be laid upon the abdomen. The patients sometimes declare that they are greatly relieved by the latter.

The question of the use of purgatives in peritonitis has of late been warmly discussed. Lawson Tait and other gynæcologists have used the saline purges with the greatest benefit in post-operation peritonitis. Theoretically it appears correct to give salines in concentrated form, which cause a rapid and profuse exosmosis of serum from the intestinal vessels, relieving the congestion and reducing the œdema, which is one important factor in causing the meteorism. It is also urged that the increased peristalsis prevents the formation of adhesions. In reading the reports of these successful cases, one is not always convinced, however, that peritonitis actually existed. Still, in cases of acute peritonitis due to extension or following operation or in septic conditions the judgment of many careful men is decidedly in favor of the use of salines. I cannot speak from personal experience on this question. The majority of cases of peritonitis which come under the care of the physician follow lesions of the abdominal viscera or are due to perforation of ulcer of the stomach, the ileum, or the appendix. In such cases, particularly in the large group of appendix cases, to give saline purgatives is, to say the least, most injudicious treatment. The safety of the patient lies in the restriction of the peristalsis and the localization of the inflammation, for which purpose opium alone is of service. In these instances rectal injections should be employed to relieve the large bowel. No symptom in acute peritonitis is more serious than the tympanites, and none is more difficult to meet. The use of the long tube and injections containing turpentine may be tried. Drugs by the mouth cannot be retained.

For the vomiting, ice and small quantities of soda water may be employed. The patient should be fed on milk, but if the vomiting is distressing it is best not to attempt to give food by the mouth, but to use small nutrient enemata. In all cases of peritonitis it is best to have a surgeon in consultation early in the disease, as the question of operation may come up at any moment. I have already mentioned the conditions under which laparotomy is indicated in perforative appendicitis. The acute purulent cases, particularly those in which the streptococci occur, usually die; but the results of operative interference even in this form are steadily improving. In the acute forms of tuberculous peritonitis operative measures appear to be more hopeful, but they are not always successful.

(*b*) **Chronic Peritonitis.**—For the cases of chronic proliferative peritonitis very little can be done. The treatment is practically that of ascites. In all these forms, when the distention becomes extreme, tapping is indicated. The treatment of tuberculous peritonitis has fallen largely into

Mucous Tubercles.

~~Dr. Cummin has recorded a case reported by~~ Francis Richardson.
recorded at St. [illegible] appendicular (Mesenteric)
found the symptoms the drop enlarged, the sentence
glands which were removed. « the boy remains
well 5 years after the operation (Phil. Trans. for 1840 ii

the hands of the surgeons, and the results in many cases are very good. According to the statistics of Maurange,* of 71 cases, 28 survived the operation for more than a year. Of 26 additional cases which I have collected,† 14 were dead at the time of the report. Within two years and three months there were 6 operations performed at the Johns Hopkins Hospital in tuberculous peritonitis, with 4 recoveries.

(*c*) **Ascites.**—The treatment depends somewhat on the nature of the case. In cirrhosis early and repeated tapping may give time for the establishment of the collateral circulation, and temporary cures have followed this precedure. Permanent drainage with Southey's tube, incision, and washing out the peritonæum have also been practised. In the ascites of cardiac and renal disease the cathartics are most satisfactory, particularly the bitartrate of potash, given alone or with jalap, and the large doses of salts given an hour before breakfast with as little water as possible. These sometimes cause rapid disappearance of the effusion, but they are not so successful in ascites as in pleurisy with effusion. The stronger cathartics may sometimes be necessary. The ascites forming part of the general anasarca of Bright's disease will receive consideration under another section.

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* Paris Thesis, 1889.

† On Tuberculous Peritonitis, Johns Hopkins Hospital Reports, 1890.

SECTION VI.

DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

1. ACUTE CORYZA.

Acute catarrhal inflammation of the upper air-passages, popularly known as a "catarrh" or a "cold," is usually an independent affection, but may precede the development of another disease.

Etiology.—It prevails most extensively in the changeable weather of the spring and early winter, and may occur in epidemic form, many cases developing in a community within a few weeks. These outbreaks are very like, though less intense than the epidemic influenza, cases of which may begin with symptoms of ordinary coryza. The disease probably depends upon a micro-organism. Irritating fumes, such as those of iodine or ammonia, also may cause an acute catarrh of the nose.

Symptoms.—The patient feels indisposed, perhaps chilly, has slight headache, and sneezes frequently. In severe cases there are pains in the back and limbs. There is usually slight fever, the temperature rising to 101°. The pulse is quick, the skin is dry, and there are all the features of a feverish attack. At first the mucous membrane of the nose is swollen, "stuffed up," and the patient has to breathe through the mouth. A thin, clear, irritating secretion flows, and makes the edges of the nostrils sore. The mucous membrane of the tear-ducts is swollen, so that the eyes weep and the conjunctivæ are injected. The sense of smell and, in part, the sense of taste is lost. With the nasal catarrh there is slight soreness of the throat and stiffness of the neck; the pharynx looks red and swollen, and sometimes the act of swallowing is painful. The larynx also may be involved, and the voice becomes husky or is even lost. If the inflammation extends to the Eustachian tubes there may be impairment of the hearing. In more severe cases there are bronchial irritation and cough. Occasionally there is an outbreak of labial or nasal herpes. Usually within thirty-six hours the nasal secretion becomes turbid and more profuse, the swelling of the mucosa subsides, the patient gradually becomes able to breathe through the nostrils, and within four or five days the symptoms disappear, with the exception of the increased discharge from the nose

and upper pharynx. There are rarely any bad effects from a simple coryza. When the attacks are frequently repeated the disease may become chronic.

The *diagnosis* is always easy, but caution must be exercised lest the initial catarrh of measles or severe influenza should be mistaken for the simple coryza.

Treatment.—Many cases are so mild that the patients are able to be about and to attend to their work. If there are fever and constitutional disturbance, the patient should be kept in bed and should take a simple fever mixture, and at night a drink of hot lemonade and a full dose of Dover's powder. Many persons find great benefit from the Turkish bath. For the distressing sense of tightness and pain over the frontal sinuses, cocaine is very useful and sometimes gives immediate relief. The 4-per-cent solution may be injected into the nostrils, or cotton-wool soaked in it may be inserted into them. Later, the snuff recommended by Ferrier is advantageous, composed, as it is, of morphia (gr. ij), bismuth (℥ iv), acacia powder (℥ ij). This may occasionally be blown or snuffed into the nostrils. The fluid extract of hamamelis, "snuffed" from the hand every two or three hours, is much better.

II. CHRONIC NASAL CATARRH.

(*Rhinitis; Rhinitis hypertrophica; Rhinitis atrophica*).

In *simple chronic catarrh* there is increased irritability of the mucous membrane, particularly of the erectile tissue on the septum and turbinated bones. There is a tendency to frequent stoppage of one or both nostrils and the patient very easily catches cold. The secretion is at first clear and afterward thick and tenacious. The sense of smell is not specially disturbed at this stage. With the mirror the mucous membrane looks congested and swollen and the veins may be distended.

In *hypertrophic rhinitis*, which is usually a sequel of the former condition, the nasal passages are obstructed, chiefly by enlargement of the lower turbinated bodies and swelling of the mucous membrane of the septum. Very often there is hypertrophy of the adenoid tissue in the vault of the pharynx and of the mucous membrane about the orifices of the Eustachian tubes. The two conditions frequently go together as expressed in the designation, chronic naso-pharyngeal catarrh. The symptoms of this hypertrophic rhinitis may be local or general.

The most important local symptom is the obstruction of the passage of air through the nostrils, so that the patients become mouth-breathers. During the day this may not be very distressing, but at night the mouth and throat get extremely dry and the sleep is disturbed. The voice becomes nasal in quality and in advanced cases, when the Eustachian tubes are obstructed, there may be deafness. It should ever be borne in mind by the practitioner that a very large proportion of all cases of deafness originate in chronic naso-pharyngeal catarrh. The general symptoms have been considered more fully under chronic pharyngeal catarrh and mouth-breathing.

Atrophic rhinitis, which is also known under the names coryza fetida and ozæna, may be a sequence of the hypertrophic form. Ozæna is only a symptom, and is met with in many ulcerative conditions of the nostrils, particularly as a result of syphilis, foreign bodies, caries and necrosis of the bones, and glanders. Fortunately, the atrophic form by no means necessarily follows the hypertrophic stage. The cases are much more frequent in women than in men, and usually occur early in life. The mucous membrane is thin and covered with grayish crusts which, when removed, show a slightly excoriated surface, but true ulcers are rarely seen. The erectile tissue is completely atrophied by a process of slow connective-tissue growth, or, as J. N. Mackenzie calls it, a cirrhosis. The mucous membrane of the pharynx is usually dry and glazed.

The symptoms are most distinctive, owing to the horrible odor which comes from the nose, and of which, fortunately, the patient is himself unconscious, because the sense of smell is lost. The secretion, which is puriform, dries and forms large crusts, which are dislodged by picking or which gradually fall off. The cause of the offensive odor has been much discussed—whether it is due to a special organism or to specially favorable conditions for the growth and development of the germs of putrefaction. Probably the latter view is correct.

The *treatment* of hypertrophic rhinitis consists in the thorough cleansing of the nasal passages, the removal of the pharyngeal growths, and the reduction of the hypertrophied nasal mucosa. It is best to use a simple douche, in order to keep the membrane absolutely clean. The Birmingham nasal douche is the most simple and satisfactory, and may be filled with alkaline and antiseptic or deodorizing solutions. One of the most satisfactory is the bicarbonate of soda (1½ drachm), listerine (6 drachms), and water (1 ounce). Operative procedures are necessary in a majority of the cases, and the practitioner should early call to his assistance the specialist. It is sad to think of the misery which has been entailed upon thousands of people owing to neglect of naso-pharyngeal catarrh by parents and physicians.

The treatment of atrophic rhinitis comes more properly under the special monographs.

III. AUTUMNAL CATARRH (*Hay Fever*).

An affection of the upper air-passages, often associated with asthmatic attacks, due to the action of certain stimuli upon a hypersensitive mucous membrane.

This affection was first described in 1819 by Bostock, who called it *catarrhus æstivus*. Morrill Wyman, of Cambridge, Mass., wrote a monograph on the subject, and described two forms, the "June cold," or "rose cold," which comes on in the spring, and the autumnal form which, in this country, does not develop until August and September, and never persists after a severe frost. Blakely studied its connection with the pollen of various grasses and flowers. The late George M. Beard made many

careful observations on the disease. Until recently this form of catarrh was believed to result exclusively from the action of certain irritants on the mucous membrane of the nose, particularly the pollen of plants, which, as the experiments of Blakeley showed, play an important *rôle* in the disease. Other emanations also may induce an attack, as in the case of the late Austin Flint, who was liable to coryza, or even asthma, if he slept on a certain sort of feather pillow. This, however, is only one factor in the disease. A second, most important one, was discovered in the condition of the nasal mucous membrane in these cases. Voltolini, of Breslau, in 1871, observed the cure of a case of asthma by the removal of a nasal polypus. Since that date the observations of Hack, in Germany, and particularly of Daly, of Pittsburg, Roe, of Rochester, John N. Mackenzie, of Baltimore, and Harrison Allen, of Philadelphia, have demonstrated the association of asthmatic attacks with nasal disease. Daly discovered that in a large proportion of the cases of hay asthma there was local disease of the mucous membrane of the nose, the cure of which rendered the patient insusceptible to conditions previously exciting the attacks. This has been abundantly confirmed. Still identical lesions exist in many people who never suffer with the disease, so that there must be a third factor, a neurotic constitution. In the etiology of hay fever, then, these three elements prevail—a nervous constitution, an irritable nasal mucosa, and the stimulus.

The disease affects certain families, particularly, it is said, those with a neurotic taint. The peculiarity may occur through several generations. It is certainly more common in the United States than in Europe, and much more common in the United States than in Canada. The United States Hay Fever Association now numbers thousands of members.

Dwellers in cities are more subject than residents in the country. The structural peculiarities of the nasal mucous membrane are those of hypertrophic rhinitis. Harrison Allen states that the inferior turbinated bones lie well above the floor of the nostrils, which renders the mucous membrane more liable to irritation from inhaled substances. Deflection of the septum, hypertrophy of the soft parts, and excessive hyperæsthesia, so that the mere touch with a probe may be sufficient to induce an attack, are common conditions.

Symptoms.—These are, in a majority of the cases, very like those of ordinary coryza. There may, however, be much more headache and distress, and some patients become very low-spirited. Cough is a common symptom and may be very distressing. Paroxysms of asthma may develop, so like as to be indistinguishable from the ordinary bronchial form. The two conditions may indeed alternate, the patient having at one time an attack of common hay fever and at another, under similar circumstances, an attack of bronchial asthma. Of the immediate exciting causes of the attack, unquestionably in a majority of the cases coming on in the autumn there is an association with the presence of pollen in the atmosphere, but this is only one of a host of exciting causes. In certain persons the paroxysms may develop at any season from sudden changes in the temperature. An attack may even come on through association of ideas. The well-

known experiment of J. N. Mackenzie, of inducing an attack in a susceptible person by offering her an artificial rose to smell, strikingly illustrates the neurotic element in the disease.

Treatment.—This may be comprised under three heads: First, since the disease appears in many instances to be a form of chronic neurosis, remedies which improve the stability of the nervous system may be employed—such as arsenic, phosphorus, and strychnia. Second, climatic. Dwellers in the cities of the Atlantic seaboard and of the Central States enjoy complete immunity in the Adirondacks and White Mountains. As a rule the disease is aggravated by residence in agricultural districts. The dry mountain air is unquestionably the best; there are cases, however, which do well at the seaside. Third, the thorough local treatment of the nose, particularly the destruction of the vessels and sinuses over the sensitive areas.

IV. EPISTAXIS.

Etiology.—Bleeding from the nose may result from local or constitutional conditions. Among local causes may be mentioned traumatism, small ulcers, picking or scratching the nose, new growths, and the presence of foreign bodies. In chronic nasal catarrh bleeding is not infrequent. The blood may come from one or both nostrils. The flow may be profuse after an injury.

Among general conditions with which nose-bleeding is associated, the following are the most important: It occurs with great frequency in growing children, particularly about the age of puberty; more frequently in the delicate than in the strong and vigorous. I have seen two cases of chronic recurring epistaxis in adults associated with remarkable telangiectases of the skin and visible mucous membranes.

Epistaxis is a very common event in persons of so-called plethoric habit. It is stated sometimes to precede, or to indicate a liability to, apoplexy, but this is very doubtful.

In venous engorgement, due to heart or pulmonary disease, epistaxis is not common and there may be a most extreme grade of cyanosis without its occurrence. In balloon and mountain ascensions, in the very rarefied atmosphere, hæmorrhage from the nose is a common event. In hæmophilia the nose ranks first of the mucous membranes from which bleeding arises. It occurs in all forms of chronic anæmias. It precedes the onset of certain fevers, more particularly typhoid, with which it seems associated in a special manner. Vicarious epistaxis has been described in cases of suppression of the menses. Lastly, it is said to be brought on by certain psychical impressions, but the observations on this point are not trustworthy. The blood in epistaxis results from capillary oozing or diapedesis. The mucous membrane is deeply congested and there may be small ecchymoses. The bleeding area is usually in the respiratory portion of one nostril and upon the cartilaginous septum.

Symptoms.—Slight hæmorrhage is not associated with any special features. When the bleeding is protracted the patients have the more

serious manifestations of loss of blood. In the slow dripping which takes place in some instances of hæmophilia, there may be formed a remarkable blood tumor projecting from one nostril and extending even below the mouth.

Death from ordinary epistaxis is very rare. The more blood is lost, the greater is the tendency to clotting with spontaneous cessation of the bleeding.

The *diagnosis* is usually easy. One point only need be mentioned; namely, that bleeding from the posterior nares occasionally occurs during sleep and the blood trickles into the pharynx and may be swallowed. If vomited, it may be confounded with hæmatemesis; or, if coughed up, with hæmoptysis.

Treatment.—In a majority of the cases the bleeding ceases of itself. Various simple measures may be employed, such as holding the arms above the head, the application of ice to the nose, or the injection of cold or hot water into the nostrils. Astringents, such as zinc, alum, or tannin, may be used; and the old-fashioned and sometimes successful remedy, a cobweb, may be introduced into the nostrils. If the bleeding comes from an ulcerated surface, an attempt should be made to apply chromic acid or to cauterize. If the bleeding is at all severe and obstinate, the posterior nares should be plugged. Ergot may be given internally or hypodermically. The inhalation of carbonic-acid gas may be tried or a solution of gelatine injected into the nostril.

II. DISEASES OF THE LARYNX.

I. ACUTE CATARRHAL LARYNGITIS.

This may come on as an independent affection or in association with general catarrh of the upper respiratory passages.

Etiology.—Many cases are due to catching cold or to overuse of the voice; others develop in consequence of the inhalation of irritating gases. It may occur in the general catarrh associated with influenza and measles. Very severe laryngitis is excited by traumatism, either injuries from without or the lodgment of foreign bodies. It may be caused by the action of very hot liquids or corrosive poisons.

Symptoms.—There is a sense of tickling referred to the larynx; the cold air irritates and, owing to the increased sensibility of the mucous membrane, the act of inspiration may be painful. There is a dry cough, and the voice is altered. At first it is simply husky, but soon phonation becomes painful, and finally the voice may be completely lost. In adults the respirations are not increased in frequency, but in children dyspnœa is not uncommon and may occur in spasmodic attacks. If much œdema accompanies the inflammatory swelling, there may be urgent dyspnœa.

The laryngoscope shows a swollen and tumefied mucous membrane of the larynx, particularly the ary-epiglottidean folds. The vocal cords have

lost their smooth and shining appearance and are reddened and swollen. Their mobility also is greatly impaired, owing to the infiltration of the adjoining mucous membrane and of the muscles. A slight mucoid exudation covers the parts. The constitutional symptoms are not severe. There is rarely much fever, and in many cases the patient is not seriously ill. Occasionally cases come on with greater intensity, the cough is very distressing, deglutition is painful, and there may be urgent dyspnœa.

Diagnosis.—There is rarely any difficulty in determining the nature of a case if a satisfactory laryngoscopic examination can be made. The severer forms may simulate œdema of the glottis. When the loss of voice is marked, the case may be mistaken for one of nervous aphonia, but the laryngoscope would decide the question at once. Much more difficult is the diagnosis of acute laryngitis in children, particularly in the very young, in whom it is so hard to make a proper examination. From ordinary laryngismus it is to be distinguished by the presence of fever, the mode of onset, and particularly the coryza and the previous symptoms of hoarseness or loss of voice. Membranous laryngitis may at first be quite impossible to differentiate, but in a majority of cases of this affection there are patches on the pharynx and early swelling of the cervical glands. The symptoms, too, are much more severe.

Treatment.—Rest of the larynx should be enjoined, so far as phonation is concerned. In cases of any severity the patient should be kept in bed. The room should be at an even temperature and the air saturated with moisture. Early in the disease, if there is much fever, aconite and citrate of potash may be given, and for the irritating painful cough a full dose of Dover's powder at night. An ice-bag externally often gives great relief.

II. CHRONIC LARYNGITIS.

Etiology.—The cases usually follow repeated acute attacks. The most common causes are overuse of the voice, particularly in persons whose occupation necessitates shouting in the open air. The constant inhalation of irritating substances, as tobacco-smoke, may also cause it.

Symptoms.—The voice is usually hoarse and rough and in severe cases may be almost lost. There is usually very little pain; only the unpleasant sense of tickling in the larynx, which causes a frequent desire to cough. With the laryngoscope the mucous membrane looks swollen, but much less red than in the acute condition. In association with the granular pharyngitis, the mucous glands of the epiglottis and of the ventricles may be involved.

Treatment.—The nostrils should be carefully examined, since in some instances chronic laryngitis is associated with and even dependent upon obstruction to the free passage of air through the nose. Local application must be made directly to the larynx, either with a brush or by means of a spray. Among the remedies most recommended are the solutions of nitrate of silver, chlorate of potash, perchloride of zinc, and tannic acid. Insufflations of bismuth are sometimes useful.

Among directions to be given are the avoidance of heated rooms and loud speaking, and abstinence from tobacco and alcohol. The throat should not be too much muffled, and morning and evening the neck should be sponged with cold water.

III. ŒDEMATOUS LARYNGITIS.

Etiology.—Œdema of the glottis, or, more correctly, of the structures which form the glottis, is a very serious affection which is met with (*a*) As a rare sequence of ordinary acute laryngitis. (*b*) In chronic diseases of the larynx, as syphilis or tubercle. (*c*) In severe inflammatory diseases like diphtheria, in erysipelas of the neck, and in various forms of cellulitis. (*d*) Occasionally in the acute infectious diseases—scarlet fever, typhus, or typhoid. In Bright's disease, either acute or chronic, there may be a rapidly developing œdema. (*e*) In angio-neurotic œdema.

Symptoms.—There is dyspnœa, increasing in intensity, so that within an hour or two the condition becomes very serious. There is sometimes marked stridor in respiration. The voice becomes husky and disappears. The laryngoscope shows enormous swelling of the epiglottis, which can sometimes be felt with the finger or even seen when the tongue is strongly depressed with a spatula. The ary-epiglottidean folds are the seat of the chief swelling and may almost meet in the middle line. Occasionally the œdema is below the true cords.

The diagnosis is rarely difficult, inasmuch as even without the laryngoscope the swollen epiglottis can be seen or felt with the finger. The disease is very fatal.

Treatment.—An ice-bag should be placed on the larynx, and the patient given ice to suck. If the symptoms are urgent, the throat should be sprayed with a strong solution of cocaine, and the swollen epiglottis scarified. If relief does not follow, tracheotomy should immediately be performed. The high rate of mortality is due to the fact that this operation is as a rule too long delayed.

IV. SPASMODIC LARYNGITIS (*Laryngismus stridulus*).

Spasm of the glottis is met with in many affections of the larynx, but there is a special disease in children which has received the above-mentioned and other names.

Etiology.—A purely nervous affection, without any inflammatory condition of the larynx, it occurs in children between the ages of six months and three years, and is most commonly seen in connection with rickets. As Escherich has shown, the disease has close relations with tetany and may display many of the accessory phenomena of this disease. Often the attack comes on when the child has been crossed or scolded. Mothers sometimes call the attacks "passion fits" or attacks of "holding the breath." It was supposed at one time that they were associated with en-

largement of the thymus, and the condition therefore received the name of *thymic asthma.*

The actual state of the larynx during a paroxysm is a spasm of the adductors, but the precise nature of the influences causing it is not yet known, whether centric or reflex from peripheral irritation. The disease is not so common in America as in England.

Symptoms.—The attacks may come on either in the night or in the day; often just as the child awakes. There is no cough, no hoarseness, but the respiration is arrested and the child struggles for breath, the face gets congested, and then, with a sudden relaxation of the spasm, the air is drawn into the lungs with a high-pitched crowing sound, which has given to the affection the name of "child-crowing." Convulsions may occur during an attack or there may be carpo-pedal spasms. Death may, but rarely does, occur during the attack. With the cyanosis the spasm relaxes and respiration begins. The attacks may recur with great frequency throughout the day.

Treatment.—The gums should be carefully examined and, if swollen and hot, freely lanced. The bowels should be carefully regulated, and as these children are usually delicate or rickety, nourishing diet and cod-liver oil should be given. By far the most satisfactory method of treatment is the cold sponging. In severe cases, two or three times a day the child should be placed in a warm bath and the back and chest thoroughly sponged for a minute or two with cold water. Since learning this practice from Ringer, at the University Hospital, I have seen many cases in which it proved successful. It may be employed when the child is in a paroxysm, though if the attack is severe and the lividity is great it is much better to dash cold water into the face. Sometimes the introduction of the finger far back into the throat will relieve the spasm.

Spasmodic croup, believed to be a functional spasm of the muscles of the larynx, is an affection seen most commonly between the ages of two and five years. According to Trousseau's description, the child goes to bed well, and about midnight or in the early morning hours awakes with oppressed breathing, harsh, croupy cough, and perhaps some huskiness of voice. The oppression and distress for a time are very serious, the face is congested, and there are signs of approaching cyanosis. The attack passes off abruptly, the child falls asleep and awakes the next morning feeling perfectly well. These attacks may be repeated for several nights in succession, and usually cause great alarm to the parents. Whether this is entirely a functional spasm is, I think, doubtful. There are instances in which the child is somewhat hoarse throughout the day, and has slight catarrhal symptoms and a brazen, croupy cough. There is probably slight catarrhal laryngitis with it. These cases are not infrequently mistaken for true croup, and parents are sometimes unnecessarily disturbed by the serious view which the physician takes of the case. Too often the poor child, deluged with drugs, is longer in recovering from the treatment than he would be from the disease. To allay the spasm a whiff of chloroform may be administered, which will in a few moments give relief, or the child may be placed in a hot bath. A prompt emetic, such as zinc or wine of ipecac, will usually

relieve the spasm, and is specially indicated if the child has overloaded the stomach through the day.

V. TUBERCULOUS LARYNGITIS.

Etiology.—Tubercles may develop primarily in the laryngeal mucosa, but in the great majority of cases the affection is secondary to pulmonary tuberculosis, in which it is met with in a variable proportion of from 18 to 30 per cent. Laryngitis may occur very early in pulmonary tuberculosis. There may be well-marked involvement of the larynx with signs of very limited trouble at one apex. These are cases which, in my experience, run a very unfavorable course.

Morbid Anatomy.—The mucosa is at first swollen and presents scattered tubercles, which seem to begin in the neighborhood of the blood-vessels. By their fusion small tuberculous masses arise, which caseate and finally ulcerate, leaving shallow irregular losses of substance. The ulcers are usually covered with a grayish exudation, and there is a general thickening of the mucosa about them, which is particularly marked upon the arytenoids. The ulcers may erode the true cords and finally destroy them, and passing deeply may cause perichondritis with necrosis and occasionally exfoliation of the cartilages. The disease may extend laterally and involve the pharynx, and downward over the mucous membrane, covering the cricoid cartilage toward the œsophagus. Above, it may reach the posterior wall of the pharynx, and in rare cases extend to the fauces and tonsils. The epiglottis may be entirely destroyed. There are rare instances in which cicatricial changes go on to such a degree that stenosis of the larynx is induced.

Symptoms.—The first indication is slight huskiness of the voice, which finally deepens to hoarseness, and in advanced stages there may be complete loss of voice. There is something very suggestive in the early hoarseness of tuberculous laryngitis. My attention has frequently been directed to the lungs simply by the quality of the voice.

The cough is in part due to involvement of the larynx. Early in the disease it is not very troublesome, but when the ulceration is extensive it becomes husky and ineffectual. Of the symptoms of laryngeal tuberculosis, none is more aggravating than the dysphagia, which is met with particularly when the epiglottis is involved, and when the ulceration has extended to the pharynx. There is no more distressing or painful complication in phthisis. In instances in which the epiglottis is in great part destroyed, with each attempt to take food there are distressing paroxysms of cough, and even of suffocation.

With the laryngoscope there is seen early in the disease a pallor of the mucous membrane, which also looks thickened and infiltrated, particularly that covering the arytenoid cartilages. The tuberculous ulcers are very characteristic. They are broad and shallow, with gray bases and ill-defined outlines. The vocal cords are infiltrated and thickened, and ulceration is very common.

The diagnosis of tuberculous laryngitis is rarely difficult, as it is usually associated with well-marked pulmonary disease. In case of doubt some of the secretion from the base of an ulcer should be removed and examined for bacilli.

Treatment.—Physicians pay scarcely sufficient attention to the laryngeal complications of consumption. The ulcers should be sprayed and kept thoroughly cleansed. Solutions of tannic acid, nitrate of silver, or sulphide of zinc may be employed. The insufflation, two or three times a day, of a powder of iodoform, with morphia, after thoroughly cleansing the ulcers with a spray, relieves the pain in a majority of the cases. Cocaine (4-per-cent solution) applied with the atomizer will often enable the patient to swallow his food comfortably. There are, however, distressing cases of extensive laryngeal and pharyngeal ulceration in which even cocaine loses its good effects. When the epiglottis is lost the difficulty in swallowing becomes very great. Wolfenden states that this may be obviated if the patient hangs his head over the side of the bed and sucks milk through a rubber tubing from a mug placed on the floor.

VI. SYPHILITIC LARYNGITIS.

Syphilis attacks the larynx with great frequency. It may result from the inherited disease or be a secondary or tertiary manifestation of the acquired form.

Symptoms.—In secondary syphilis there is occasionally erythema of the larynx, which may go on to definite catarrh, but has nothing characteristic. The process may proceed to the formation of superficial whitish ulcers, usually symmetrically placed on the cords or ventricular bands. Mucous patches and condylomata are rarely seen. The symptoms are practically those of slight loss of voice with laryngeal irritation, as in the simple catarrhal form.

The tertiary laryngeal lesions are numerous and very serious. True gummata, varying in size from the head of a pin to a small nut, develop in the submucous tissue, most commonly at the base of the epiglottis. They go through the changes characteristic of these structures and may either break down, producing extensive and deep ulceration, or—and this is more characteristic of syphilitic laryngitis—in their healing form a fibrous tissue which shrinks and produces stenosis. The ulceration is apt to extend deeply and involve the cartilage, inducing necrosis and exfoliation, and even hæmorrhage from erosion of the arteries. Œdema may suddenly prove fatal. The cicatrices which follow the sclerosis of the gummata or the healing of the ulcers produce great deformity. The epiglottis, for instance, may be tied down to the pharyngeal wall or to the epiglottic folds, or even to the tongue; and eventually a stenosis results, which may necessitate tracheotomy.

The laryngeal symptoms of inherited syphilis have the usual course of these lesions and appear either early, within the first five or six months, or after puberty; most commonly in the former period. Of 76 cases, J. N.

Mackenzie found that 63 occurred within the first year. The gummatous infiltration leads to ulceration, most commonly of the epiglottis and in the ventricles, and the process may extend deeply and involve the cartilage. Cicatricial contraction may also occur.

The diagnosis of syphilis of the larynx is rarely difficult, since it occurs most commonly in connection with other symptoms of the disease.

Treatment.—The administration of constitutional remedies is the most important, and under mercury and iodide of potassium the local symptoms may rapidly be relieved. The tertiary laryngeal manifestations are always serious and difficult to treat. The deep ulceration is specially hard to combat, and the cicatrization may necessitate tracheotomy, or the gradual dilatation, as practised by Schroetter.

III. DISEASES OF THE BRONCHI.

I. ACUTE BRONCHITIS.

Acute catarrhal inflammation of the bronchial mucous membrane is a very common disease, rarely serious in healthy adults, but very fatal in the old and in the young, owing to associated pulmonary complications. It is bilateral and affects either the larger and medium sized tubes or the smaller bronchi, in which case it is known as capillary bronchitis.

We shall speak only of the former, as the latter is part and parcel of broncho-pneumonia.

Etiology.—Acute bronchitis is a common sequel of catching cold, and is often nothing more than the extension downward of an ordinary coryza. It occurs most frequently in the changeable weather of early spring and late autumn. Its association with cold is well indicated by the popular expression "cold on the chest." It may prevail as an epidemic apart from influenza, of which it is an important feature.

Acute bronchitis is associated with many other affections, notably measles. It is by no means rare at the onset of typhoid fever and malaria. It is present also in asthma and whooping-cough. The subjects of spinal curvature are specially liable to the disease. The bronchitis of Bright's disease, gout, and heart-disease is usually a chronic form. It attacks persons of all ages, but most frequently the young and the old. There are individuals who have a special disposition to bronchial catarrh, and the slightest exposure is apt to bring on an attack. Persons who live an out-of-door life are usually less subject to the disease than those who follow sedentary occupations.

The affection is probably microbic, though we have as yet no definite evidence upon this point.

Morbid Anatomy.—The mucous membrane of the trachea and bronchi is reddened, congested, and covered with mucus and muco-pus, which may be seen oozing from the smaller bronchi, some of which are dilated. The finer changes in the mucosa consist in desquamation of the

ciliated epithelium, swelling and œdema of the submucosa, and infiltration of the tissue with leucocytes. The mucous glands are much swollen.

Symptoms.—The symptoms of an ordinary "cold" accompany the onset of an acute bronchitis. The coryza extends to the tubes, and may also affect the larynx, producing hoarseness, which in many cases is marked. A chill is rare, but there is invariably a sense of oppression, with heaviness and languor and pains in the bones and back. In mild cases there is scarcely any fever, but in severer forms the range is from 101° to 103°. The bronchial symptoms set in with a feeling of tightness and rawness beneath the sternum and a sensation of oppression in the chest. The cough is rough at first, and often of a ringing character. It comes on in paroxysms which rack and distress the patient extremely. During the severe spells the pain may be very intense beneath the sternum and along the attachments of the diaphragm. At first the cough is dry and the expectoration scanty and viscid, but in a few days the secretion becomes muco-purulent and abundant, and finally purulent. With the loosening of the cough great relief is experienced. The sputum is made up largely of pus-cells, with a variable number of the large round alveolar cells, many of which contain carbon grains, while others have undergone the myelin degeneration.

Physical Signs.—The respiratory movements are not greatly increased in frequency unless the fever is high. There are instances, however, in which the breathing is rapid and when the smaller tubes are involved there is dyspnœa. On palpation the bronchial fremitus may often be felt. On auscultation in the early stage, piping sibilant rales are everywhere to be heard. They are very changeable, and appear and disappear with coughing. With the relaxation of the bronchial membranes and the greater abundance of the secretion, the rales change and become mucous and bubbling in quality. The bases of the lungs should be carefully examined each day, particularly in children and the aged.

The *course* of the disease depends on the conditions under which it develops. In healthy adults, by the end of a week the fever subsides and the cough loosens. In another week or ten days convalescence is fully established. In young children the chief risk is in the extension of the process downward. In measles and whooping-cough, the ordinary bronchial catarrh is very apt to descend to the finer tubes, which become dilated and plugged with muco-pus, inducing areas of collapse, and finally bronchopneumonia. This extension is indicated by changes in the physical signs. Usually at the base the rales are subcrepitant and numerous and there may be areas of defective resonance and of feeble or distant tubular breathing. In the aged and debilitated there are similar dangers if the process extends from the larger to the smaller tubes. In old age the bronchial mucosa is less capable of expelling the mucus, which is more apt to sag to the dependent parts and induce dilatation of the tubes with extension of the inflammation to the contiguous air-cells.

The *diagnosis* of acute bronchitis is rarely difficult. Although the mode of onset may be brusque and perhaps simulate pneumonia, yet the absence of dulness and blowing breathing, and the general character of

the bronchial inflammation, render the diagnosis simple. About once a year I see a case of typhoid fever, in which the diagnosis at first has been acute bronchitis. The complication of broncho-pneumonia is indicated by the greater severity of the symptoms, particularly the dyspnœa, the changed color, and the physical signs.

Treatment.—In mild cases, household measures suffice. The hot foot-bath, or the warm bath, a drink of hot lemonade, and a mustard plaster on the chest will often give relief. For the dry, racking cough, the symptom most complained of by the patient, Dover's powder is the best remedy. It is a popular belief that quinine, in full doses, will check an oncoming cold on the chest, but this is doubtful. It is a common custom when persons feel the approach of a cold to take a Turkish bath, and though the tightness and oppression may be relieved by it, there is in a majority of the cases great risk. Some of the severest cases of bronchitis which I have seen have followed this initial Turkish bath. No doubt, if the person could go to bed directly from the bath, its action would be beneficial, but there is great risk of catching additional "cold" in going home from the bath. Relief is obtained from the unpleasant sense of rawness by keeping the air of the room saturated with moisture, and in this dry stage the old-fashioned mixture of the wines of antimony and ipecacuanha with liquor ammonii acetatis and nitrous ether is useful. If the pulse is very rapid, tincture of aconite may be given, particularly in the case of children. For the cough, when dry and irritating, opium should be freely used in the form of Dover's powder. Of course, in the very young and the aged care must be exercised in the use of opium, particularly if the secretions are free; but for the distressing, irritative cough, which keeps the patient awake, no remedy can take its place. As the cough loosens and the expectoration is more abundant, the patient becomes more comfortable. In this stage it is customary to ply him with expectorants of various sorts. Though useful occasionally, they should not be given as a matter of routine. A mixture of squills, ammonia, and senega is a favorite one with many practitioners at this stage.

In the acute bronchitis of children, if the amount of secretion is large and difficult to expectorate, or if there is dyspnœa and the color begins to get dusky, an emetic (a tablespoonful of ipecac wine) should be given at once and repeated if necessary.

II. CHRONIC BRONCHITIS.

Etiology.—This affection may follow repeated attacks of acute bronchitis, but it is most commonly met with in chronic lung affections, heart-disease, aneurism of the aorta, gout, and renal disease. It is frequent in the aged; the young rarely are affected. Climate and season have an important influence. It is the winter cough of the old man, which recurs with regularity as the weather gets cold and changeable.

Morbid Anatomy.—The bronchial mucosa presents a great variety of changes, depending somewhat upon the disease with which chronic

bronchitis is associated. In some cases the mucous membrane is very thin, so that the longitudinal bands of elastic tissue stand out prominently. The tubes are dilated, the muscular and glandular tissues are atrophied, and the epithelium is in great part shed.

In other instances the mucosa is thickened, granular, and infiltrated. There may be ulceration, particularly of the mucous follicles. Bronchial dilatations are not uncommon and emphysema is a constant accompaniment.

Symptoms.—In the form met with in old men, associated with emphysema, gout, or heart-disease, the chief symptoms are as follows: Shortness of breath, which may not be noticeable except on exertion. The patients "puff and blow" on going up hill or up a flight of stairs. This is due not so much to the chronic bronchitis itself as to associated emphysema or even to cardiac weakness. They complain of no pain. The cough is variable, changing with the weather and with the season. During the summer they may remain free, but each succeeding winter the cough comes on with severity and persists. There may be only a spell in the morning, or the chief distress is at night. The sputum in chronic bronchitis is very variable. In cases of the so-called dry catarrh there is no expectoration. Usually, however, it is abundant, muco-purulent, or distinctly purulent in character. There are instances in which the patient coughs up for years a thin fluid sputum. There is rarely fever. The general health may be good and the disease may present no serious features apart from the liability to induce emphysema and bronchiectasy. In many cases it is an incurable affection. Patients improve and the cough disappears in the summer time only to return during the winter months.

Physical Signs.—The chest is usually distended, the movements are limited, and the condition is often that which we see in emphysema. The percussion note is clear or hyperresonant. On auscultation, expiration is prolonged and wheezy and rhonchi of various sorts are heard—some high-pitched and piping, others deep-toned and snoring. Crepitation is common at the bases.

Clinical Varieties.—The description just given is of the ordinary chronic bronchitis which occurs in connection with emphysema and heart-disease and in many elderly men. There are certain forms which merit special description: (*a*) On several occasions I have met with a form of *chronic bronchitis*, particularly in women, which comes on between the ages of twenty and thirty and may continue indefinitely without serious impairment of the health.

(*b*) *Bronchorrhœa.*—Excessive bronchial secretion is met with under several conditions. It must not be mistaken for the profuse expectoration of bronchiectasy. The secretion may be very liquid and watery—*bronchorrhœa serosa*, and in extraordinary amount. More commonly, it is purulent though thin, and with greenish or yellow-green masses. It may be thick and uniform. This profuse bronchial secretion is usually a manifestation of chronic bronchitis and may lead to dilatation of the tubes and ultimately to fetid bronchitis. In the young the condition may persist for years without impairment of health and without apparently damaging the lungs.

(*c*) *Putrid Bronchitis.*—Fetid expectoration is met with in connection with bronchiectasis, gangrene, abscess, or with decomposition of secretions within phthisical cavities and in an empyema which has perforated the lung. There are instances in which, apart from any of these states, the expectoration has a fetid character. The sputa are abundant, usually thin, grayish-white in color, and they separate into an upper fluid layer capped with frothy mucus and a thick sediment in which may sometimes be found dirty yellow masses the size of peas or beans—the so-called Dittrich's plugs. The affection is very rare apart from the above-mentioned conditions. In severe cases it leads to changes in the bronchial walls, pneumonia, and often to abscess or gangrene. Metastatic brain abscess has followed putrid bronchitis in a certain number of cases.

(*d*) *Dry Catarrh.*—The *catarrhe sec* of Laennec, a not uncommon form, is characterized by paroxysms of coughing of great intensity, with little or no expectoration. It is usually met with in elderly persons with emphysema, and is one of the most obstinate of all varieties of bronchitis.

In England the damp cold of the unwarmed houses is responsible in great part for the prevalence of chronic bronchitis among the aged and weak. An equable, warm temperature is of the first importance to all persons prone to the disease.

Treatment.—By far the most satisfactory method of treating the recurring winter bronchitis is change of climate. Removal to a southern latitude may prevent the onset. Southern France, southern California, and Florida furnish winter climates in which the subjects of chronic bronchitis live with the greatest comfort. All cases of prolonged bronchial irritation are benefited by change of air.

The first endeavor in treating a case of chronic bronchitis is to ascertain, if possible, whether there are constitutional or local affections with which it is associated. In many instances the urine is found to be highly acid, perhaps slightly albuminous, and the arteries are stiff. In the form associated with this condition, sometimes called gouty bronchitis, the attacks seem related to the defective renal elimination, and to this condition the treatment should be first directed. In other instances there are heart-disease and emphysema. In the form occurring in old men much may be done in the way of prophylaxis. Septuagenarians should read Oliver Wendell Holmes's * "De Senectute" with reference to the care of the health. There is no doubt that with prudence even in our changeable winter weather much may be done to prevent the onset of chronic bronchitis. Woollen undergarments should be used and especial care should be taken in the spring months not to change them for lighter ones before the warm weather is established.

Cure is seldom effected by medicinal remedies. There are instances in which iodide of potassium acts with remarkable benefit, and it should always be given a trial in cases of paroxysmal bronchitis of obscure origin. For the morning cough, bicarbonate of sodium (gr. xv), chloride of sodium (gr. v), spirits of chloroform (ɱv) in anise water and taken with an equal

* Over the Tea-cups, Boston, 1890.

amount of warm water will be found useful (Fowler). When there is much sense of tightness and fulness of the chest, the portable Turkish bath may be tried. When the secretion is excessive muriate of ammonia and senega are useful. Stimulating expectorants are contraindicated. When the heart is feeble, the combination of digitalis and strychnia is very beneficial. Turpentine, the old-fashioned remedy so warmly recommended by the Dublin physicians, has in many quarters fallen undeservedly into disuse. Preparations of tar, creasote, and terebene are sometimes useful. Of other balsamic remedies, sandal-wood, the compound tincture of benzoin, copaiba, balsam of Peru or tolu may be used. Inhalations of eucalyptus and of the spray of ipecacuanha wine are often very useful. If fetor be present, carbolic acid in the form of spray (10 to 20 per cent solution) will lessen the odor, or thymol (1 to 1,000). For urgent dyspnœa with cyanosis, bleeding from the arm gives most relief.

III. BRONCHIECTASIS.

Etiology.—Dilatation of the bronchi occurs under the following conditions: (1) As a congenital defect or anomaly. Such cases are extremely rare, commonly unilateral. Grawitz has described the condition as *bronchiectasis universalis.* Welch has met an instance in a young girl. (2) In connection with inflammation of the bronchi, particularly when this leads to weakness of the walls with the accumulation of secretion. I have seen an instance after influenza. Under this category comes the dilatation met with in chronic bronchitis and emphysema, the dilated bronchi in chronic phthisis, in the catarrhal pneumonias of children, and particularly the dilatation which results from the presence of foreign bodies in the air-tubes or from pressure, as of an aneurism on one bronchus. (3) In extreme contraction of the lung tissue, whether due to interstitial pneumonia or to compression by pleural adhesions, bronchial dilatation is a common though not a constant accompaniment.

Unquestionably the weakening of the bronchial wall is the most important, probably the essential, factor in inducing bronchiectasy, since the wall is then not able to resist the pressure of air in severe spells of coughing and in straining. In some instances the mere weight of the accumulated secretion may be sufficient to distend the terminal tubules, as is seen in compression of a bronchus by aneurism.

Morbid Anatomy.—Two chief forms are recognized—the *cylindrical* and the *saccular*—which may exist together in the same lung. The condition may be general or partial. Universal bronchiectasis is always unilateral. It occurs in rare congenital cases and is occasionally seen as a sequence of interstitial pneumonia. The entire bronchial tree is represented by a series of sacculi opening one into the other. The walls are smooth and possibly without ulceration or erosion except in the dependent parts. The lining membrane of the sacculi is usually smooth and glistening. The dilatations may form large cysts immediately beneath the pleura. Intervening between the sacculi is a dense cirrhotic lung tissue. The

partial dilatations—the saccular and cylindrical—are common in chronic phthisis, particularly at the apex, in chronic pleurisy at the base, and in emphysema. Here the dilatation is more commonly cylindrical, sometimes fusiform. The bronchial mucous membrane is much involved and sometimes there is a narrowing of the lumen. Occasionally one meets with a single saccular bronchiectasy in connection with chronic bronchitis or emphysema. Some of these look like simple cysts, with smooth walls, without fluid contents. A form of acute bronchiectasis in children has been described by Sharkey, Carr, and others. A good account of it is given in Fowler and Godlee's work on the lungs.

Histologically the bronchi which are the seat of dilatation show important changes. In the large, smooth dilatations the cylindrical is replaced by a pavement epithelium. The muscular layer is stretched, atrophied, and the fibres separated; the elastic tissue is also much stretched and separated. In the large saccular bronchiectases and in some of the cylindrical forms, due to retained secretions, the lining membrane is ulcerated. The contents of some of the larger bronchiectatic cavities are horribly fetid.

Symptoms.—In the limited dilatations of phthisis, emphysema, and chronic bronchitis, the symptoms are in great part those of the original disease, and the condition often is not suspected during life.

In extensive saccular bronchiectasy the characters of the cough and expectoration are distinctive. The patient will pass the greater part of the day without any cough and then in a severe paroxysm will bring up a large quantity of sputum. Sometimes change of the position will bring on a violent attack, probably due to the fact that some of the secretion flows from the dilatation to a normal tube. The daily spell of coughing is usually in the morning. The expectoration is in many instances very characteristic. It is grayish or grayish brown in color, fluid, purulent, with a peculiar acid, sometimes fetid, odor. Placed in a conical glass, it separates into a thick granular layer below and a thin mucoid intervening layer above, which is capped by a brownish froth. Microscopically it consists of pus-corpuscles, often large crystals of fatty acids, which are sometimes in enormous numbers over the field and arranged in bunches. Hæmatoidin crystals are sometimes present. Elastic fibres are seldom found except when there is ulceration of the bronchial walls. Tubercle bacilli are not present. In some cases the expectoration is very fetid and has all the characters of that described under fetid bronchitis. Nummular expectoration, such as comes from phthisical cavities, is not common. Hæmorrhage occurred in 14 out of 35 cases analyzed by Fowler. Abscess of the brain has in a few instances followed the bronchiectasis. Rheumatoid affections may develop, and it is one of the conditions with which the pulmonary osteo-arthropathy is commonly associated.

The *diagnosis* is not possible in a large number of the cases. In the extensive sacculated forms, unilateral and associated with interstitial pneumonia or chronic pleurisy, the diagnosis is easy. There is contraction of the side, which in some instances is not at all extreme. The cavernous signs may be chiefly at the base and may vary according to the condi-

tion of the cavity, whether full or empty. There may be the most exquisite amphoric phenomena and loud resonant rales. The condition persists for years and is not inconsistent with a tolerably active life. The patients frequently show signs of marked embarrassment of the pulmonary circulation. There is cyanosis on exertion, the finger-tips are clubbed, and the nails incurved. A condition very difficult to distinguish from bronchiectasy is a limited pleural cavity communicating with a bronchus.

Treatment.—Medical treatment is not satisfactory, since it is impossible to heal the cavity. I have practised the injection of antiseptic fluids in some instances with benefit. Intratracheal injections have been very warmly recommended of late. With a suitable syringe a drachm may be injected twice a day of the following solution: Menthol 10 parts, guaiacol 2 parts, olive oil 88 parts. The creasote vapor bath may be given in a small room. The patient's eyes must be protected with well-fitting goggles, and the nostrils stuffed with cotton-wool. Commercial creasote is poured into a metal saucer on a tripod and the saucer heated by a spirit lamp. At first the vapor is very irritating and disagreeable, but the patient gets used to it. The bath should be taken at first every other day for fifteen minutes, then gradually increased to an hour daily. The treatment should be continued for three months. Fowler states that he has known the fetor to disappear. In suitable cases drainage of the cavities may be attempted, particularly if the patient is in fairly good condition. For the fetid secretion turpentine may be given, or terebene, and inhalations used of carbolic acid or thymol.

IV. BRONCHIAL ASTHMA.

Asthma is a term which has been applied to various conditions associated with dyspnœa—hence the names cardiac and renal asthma—but its use should be limited to the affection known as bronchial or spasmodic asthma.

Etiology.—All writers agree that there is in a majority of cases of bronchial asthma a strong neurotic element. Many regard it as a neurosis in which, according to one view, spasm of the bronchial muscles, according to the other turgescence of the mucosa, results from disturbed innervation, pneumogastric or vaso-motor. Of the numerous theories the following are the most important:

(1) That it is due to spasm of the bronchial muscles, a theory which has perhaps the largest number of adherents. The original experiments of C. J. B. Williams, upon which it is largely based, have not, however, been confirmed of late years.

(2) That the attack is due to swelling of the bronchial mucous membrane—fluctionary hyperæmia (Traube), vaso-motor turgescence (Weber), diffuse hyperæmic swelling (Clark).

(3) That in many cases it is a special form of inflammation of the smaller bronchioles—*bronchiolitis exudativa* (Curschmann). Other theo-

ries which may be mentioned are that the attack depends on spasm of the diaphragm or on reflex spasm of all the inspiratory muscles.

As already mentioned, the so-called hay fever is an affection which has many resemblances to bronchial asthma, with which the attacks may alternate. In the suddenness of onset and in many of their features these diseases have the same origin and differ only in site, as suggested by Sir Andrew Clark and now generally acknowledged by specialists. Making due allowance for anatomical differences, if the structural changes occurring in the nasal mucous membrane during an attack of hay fever were to occur also in various parts of the bronchial mucosa, their presence there would afford a complete and adequate explanation of the facts observed during a paroxysm of bronchial asthma (Clark). With this statement I fully agree, but the observations of Curschmann have directed attention to a feature in asthma which has been neglected; namely, that in a majority of the cases it is associated with an exudation, such as might be supposed to come from a turgescent mucosa and which is of a very characteristic and peculiar character. The hyperæmia and swelling of the mucosa and the extremely viscid, tenacious mucus explain well the hindrance to inspiration and expiration and also the quality of the rales. An œdema of the angio-neurotic type has been described in the hands and arms in asthma (J. S. Billings, Jr.).

Some general facts with reference to etiology may be mentioned. The affection sometimes runs in families, particularly those with irritable and unstable nervous systems. The attack may be associated with neuralgia or, as Salter mentions, even alternate with epilepsy. Men are more frequently affected than women. The disease often begins in childhood and sometimes lasts until old age. It may follow an attack of whooping-cough. One of its most striking peculiarities is the *bizarre* and extraordinary variety of circumstances which at times induce a paroxysm. Among these local conditions climate or atmosphere are most important. A person may be free in the city and invariably suffer from an attack when he goes into the country, or into one special part of the country. Such cases are by no means uncommon. Breathing the air of a particular room or a dusty atmosphere may bring on an attack. Odors, particularly of flowers and of hay, or emanations from animals, as the horse, dog, or cat, may at once cause an outbreak. Fright or violent emotion of any sort may bring on a paroxysm. Uterine and ovarian troubles were formerly thought to induce attacks and may do so in rare instances. Diet, too, has an important influence, and in persons subject to the disease severe paroxysms may be induced by overloading the stomach, or by taking certain articles of food. Chronic cases, in which the attacks recur year after year, gradually become associated with emphysema, and every fresh "cold" induces a paroxysm. And lastly, many cases of bronchial asthma are associated with affections of the nose, particularly with hypertrophic rhinitis and nasal polypi. According to some specialists of large experience, all cases of bronchial asthma have some affection of the upper air-passages, but I am convinced from personal observation that this is erroneous. Still physicians must acknowledge the debt which we owe to Voltolini, Hack, Daly, Roe, and others who have

shown the close connection which exists between affections of the naso-pharynx and many cases of bronchial asthma.

Briefly stated then, bronchial asthma is a neurotic affection, characterized by hyperæmia and turgescence of the mucosa of the smaller bronchial tubes and a peculiar exudate of mucin. The attacks may be due to direct irritation of the bronchial mucosa or may be induced reflexly, by irritation of the nasal mucosa, and indirectly, too, by reflex influences, from stomach, intestines, or genital organs.

Symptoms.—Premonitory sensations precede some attacks, such as chilly feelings, a sense of tightness in the chest, flatulence, the passage of a large quantity of urine, or great depression of spirits. Nocturnal attacks are common. After a few hours' sleep, the patient is aroused with a distressing sense of want of breath and a feeling of great oppression in the chest. Soon the respiratory efforts become violent, all the accessory muscles are brought into play, and in a few minutes the patient is in a paroxysm of the most intense dyspnœa. The face is pale, the expression anxious, speech is impossible, and in spite of the most strenuous inspiratory efforts very little air enters the lungs. Expiration is prolonged and also wheezy. The number of respirations, however, is not much increased. The asthmatic fit may last from a few minutes to several hours. When severe, the signs of defective aëration soon appear, the face becomes bedewed with sweat, the pulse is small and quick, the extremities get cold, and just as the patient seems to be at his worst, the breathing begins to get easier, and often with a paroxysm of coughing relief is obtained and he sinks exhausted to sleep. The relief may be but temporary and a second attack may soon come on. In a majority of the cases even in the intervals between the asthmatic fits the respiration is somewhat embarrassed. The cough is at first very tight and dry and the expectoration is expelled with the greatest difficulty.

The physical signs during an attack are very characteristic. On inspection the thorax looks enlarged, barrel-shaped, and is fixed, the amount of expansion being altogether disproportionate to the intensity of the inspiratory movements. The diaphragm is lowered and moves but slightly. Inspiration is short and quick, expiration prolonged. Percussion may not reveal any special difference, but there is sometimes marked hyperresonance, particularly in cases which have had repeated attacks.

On auscultation, with inspiration and expiration, there are innumerable sibilant and sonorous rales of all varieties, piping and high-pitched, low-pitched and grave. Later in the attack there are moist rales.

The *sputum* in bronchial asthma is quite distinctive, unlike that which occurs in any other affection. Early in the attack it is brought up with great difficulty and is in the form of rounded gelatinous masses, the so-called "*perles*" of Laennec. Though ball-like, they can be unfolded and really represent moulds in mucus of the smaller tubes. The entire expectoration may be made up of these somewhat translucent-looking pellets, floating in a small quantity of thin mucus. Some of them are opaque. Often with a naked eye a twisted spiral character can be seen, particularly if the sputum is spread on a glass with a black background. Microscopic-

Emphysema of the neck may develop during violent attacks of coughing

(Med Surgical Rep. Ward C. Morris 1848

ally, many of these pellets have a spiral structure, which renders them among the most remarkable bodies met with in sputum. It is not a little curious that they should have been practically overlooked until described a few years ago by Curschmann. Under the microscope the spirals are of two forms. In one there is simply a twisted, spirally arranged mucin, in which are entangled leucocytes, the majority of which are eosinophiles. The twist may be loose or tight. The second form is much more peculiar. In the centre of a tightly coiled skein of mucin fibrils with a few scattered cells is a filament of extraordinary clearness and translucency, probably composed of transformed mucin. As Curschmann suggests, these spirals are doubtless formed in the finer bronchioles and constitute the product of an acute bronchiolitis. It is difficult to explain their spiral nature. I do not know of any observations upon the course of the currents produced by the ciliated epithelium in the bronchi, but it is quite possible that their action may be rotatory, in which case, particularly when combined with spasm of the bronchial muscles, it is possible to conceive that the mucus formed in the tube might be compelled to assume a spiral form. Within two or three days the sputum changes entirely in character; it becomes muco-purulent and Curschmann's spirals are no longer to be found. They occur in all instances of true bronchial asthma in the early period of the attack. I have never seen the true spirals either in bronchitis or pneumonia. There are, in addition, in many cases, the pointed, octahedral crystals described by Leyden and sometimes called asthma crystals. They are identical with the crystals found in the semen and in the blood in leukæmia. At one time they were supposed, by their irritating character, to induce the paroxysms. Eosinophiles in the blood are enormously increased in asthma—to 25 or 35 per cent of the leucocytes, or even to 53.6 per cent in one case (J. S. Billings, Jr.).

The *course* of the disease is very variable. In severe attacks the paroxysms recur for three or four nights or even more, and in the intervals and during the day there may be wheezing and cough. Early in the disease the patient may be free in the morning, without cough or much distress, and the attacks may appear at first to be of a purely nervous character. In the long-standing cases emphysema almost invariably develops, and while the pure asthmatic fits diminish in frequency the chronic bronchitis and shortness of breath become aggravated.

We have no knowledge of the morbid anatomy of true asthma. Death during the attack is unknown. In long-standing cases the lesions are those of chronic bronchitis and emphysema.

Treatment.—The asthmatic attack usually demands immediate and prompt treatment, and remedies should be administered which experience has shown are capable of relieving the condition of the bronchial mucosa. A few whiffs of chloroform will produce prompt though temporary relaxation. In a child with very severe attacks, resisting all the usual remedies, the treatment by chloroform gave immediate and finally permanent relief. Hypodermic injections of pilocarpin (gr. $\frac{1}{8}$) will sometimes relax the mucosa in the profuse sweating. Perles of nitrite of amyl may be broken on the handkerchief or from two to five drops of the solution may be placed

upon cotton-wool and inhaled. Strong stimulants given hot or a dose of spirits of chloroform in hot whisky will sometimes induce relaxation. More permanent relief is given by the hypodermic injection of morphia or of morphia and cocaine combined. In obstinate and repeatedly recurring attacks this has proved a very satisfactory plan. The sedative antispasmodics, such as belladonna, henbane, stramonium, and lobelia, may be given in solution or used in the form of cigarettes. Nearly all the popular remedies either in this form or in pastilles contain some plant of the order *solanaceæ*, with nitrate or chlorate of potash. Excellent cigarettes are now manufactured and asthmatics try various sorts, since one form benefits one patient, another form another patient. Nitre paper made with a strong solution of nitrate of potash is very serviceable. Filling the room with the fumes of this paper prior to retiring will sometimes ward off a nocturnal attack. I have known several patients to whom tobacco smoke inhaled was quite as potent as the prepared cigarettes.

The use of compressed air in the pneumatic cabinet is very beneficial; oxygen inhalations may also be tried. In preventing the recurrence of the attacks there is no remedy so useful as iodide of potassium, which sometimes acts like a specific. From 10 to 20 grains three times a day is usually sufficient.

Particular attention should be paid to the diet of asthmatic patients. A rule which experience generally compels them to make is to take the heavy meals in the early part of the day and not retire to bed before gastric digestion is completed. As the attacks are often induced by flatulency, the carbohydrates should be restricted. Coffee is a more suitable drink than tea. In respect to climate it is very difficult to lay down rules for asthmatics. The patients are often much better in the city than in the country. The high and dry altitudes are certainly more beneficial than the sea-shore; but in protracted cases, with emphysema as a secondary complication, the rarefied air of high altitudes is not advantageous. In young persons I have known a residence for six months in Florida or southern California to be followed by prolonged freedom from attacks.

V. FIBRINOUS BRONCHITIS.

An acute or chronic affection, characterized by the formation in certain of the bronchial tubes of fibrinous casts, which are expelled in paroxysms of dyspnœa and cough.

In several diseases fibrinous moulds of the bronchi are formed. as in diphtheria and croup (with extension into the trachea and bronchi), in pneumonia, and occasionally in phthisis—conditions which, however, have nothing to do with true fibrinous bronchitis. These casts are not to be confounded with the blood-casts which occur occasionally in hæmoptysis.

Etiology.—Nothing is known of its causation. It occurs more frequently in males. It is met with at all periods of life, but is more common between the ages of twenty and forty. It has been known to attack several members of the same family. Instances have been described occurring

together as if due to some endemic influence (Pichini). The cases are rare, particularly in hospital practice. The attacks occur most commonly in the spring months. An association with tuberculosis has been frequently noted. Model, in an article from Bäumler's clinic, states that tuberculosis was present in ten of twenty-one post mortems. It has been met with also in connection with skin-diseases, such as pemphigus, impetigo, and herpes. The attacks appeared to be related in some cases to the menstrual period. Several instances have been described with heart-disease, but it seems probable that in all these conditions the connection was not causal.

Symptoms.—Acute cases are rare. They may set in with high fever, rigors, severe paroxysms of cough, and perhaps with hæmoptysis. The clinical picture resembles that of acute bronchitis, and only the expulsion of the membranous casts gives the characteristic features to the case. It is much more serious than the chronic form and fatal termination is not uncommon. N. S. Davis has reported two fatal cases. In some of the acute cases there has been affection of the tonsils, and it is possible that the disease may have been truly diphtheritic in character and due to extension of the membrane into the trachea and bronchi. The casts in these cases are not only more extensive, but they also do not present the laminated structure characteristic of true plastic bronchitis.

A patient may have a single attack without any recurrence, but in the chronic form the attacks come on at varying intervals and the disease may last for ten or even twenty years. Instances are on record in which the paroxysms have occurred at definite intervals for many months. The attacks may recur weekly or a period of a year or more may intervene. The onset is marked by bronchitic symptoms, not necessarily with fever. The cough becomes distressing and paroxysmal in character; the sputa may be blood-stained and the patient brings up rounded, ball-like masses, which, when disentangled, are found to be moulds of bronchi; the hæmorrhage may be profuse. In one of the two cases which I have seen it invariably accompanied the attack, and the whitish dendritic casts of the tubes were always entangled in the blood and clots. Urgent dyspnœa and cyanosis may be present in severe attacks. The *physical* signs are those of a severe bronchitis. It may occasionally be possible to determine the weakened or suppressed breath sounds in the affected territory and there may be deficient expansion or even retraction of the chest wall in a corresponding area, but this is in reality very difficult, and twice prior to the expulsion of the casts I failed to determine by physical examination the affected region.

As mentioned, the casts are usually rolled up and mixed with mucus or blood. When unravelled in water they present a complete mould of a secondary or tertiary bronchus with its ramifications. The size of the cast may vary with different attacks, but, as has often been noticed, the form and size may be identical at each attack as if precisely the same bronchial area was involved each time. The casts are hollow, laminated, the size of the lumen varying with the number and thickness of the laminæ. Sometimes they are almost solid. Transverse sections show a beautiful concentric arrangement. The casts have been determined by Grandy to be composed of mucus and not of fibrin. He regards the process as analogous to

the mucous colitis. The mucin appears in places to retain its fibrillary structure; in others, as in diphtheritic membrane, it has undergone the hyaline transformation. Leucocytes are imbedded in the meshes. In the centre, particularly in the smaller casts, it is not uncommon to see alveolar epithelium with numerous carbon particles. Leyden's crystals are sometimes found and occasionally Curschmann's spirals.

The pathology of the disease is obscure. The membrane is identical with that to which the term croupous is applied, and the obscurity relates not so much to the mechanism of the production, which is probably the same as in other mucous surfaces, as to the curious limitation of the affection to certain bronchial territories and the remarkable recurrence at stated or irregular intervals throughout a period of many years.

In the acute cases the *treatment* should be that of ordinary acute bronchitis. We know of nothing which can prevent the recurrence of the attacks in the chronic form. In the uncomplicated cases there is rarely any danger during the paroxysm, even though the symptoms may be most distressing and the dyspnœa and cough very severe. Inhalations of ether, steam, or atomized lime-water aid in the separation of the membranes. Pilocarpine might be useful, as in some instances it increases the bronchial secretion. The employment of emetics may be necessary, and in some cases they are effective in promoting the removal of the casts.

IV. DISEASES OF THE LUNGS.

I. CIRCULATORY DISTURBANCES IN THE LUNGS.

Congestion.—There are two forms of congestion of the lungs—active and passive.

(1) *Active Congestion of the Lungs.*—Much doubt and confusion still exist on this subject. French writers, following Woillez, regard it as an independent primary affection (*maladie de Woillez*), and in their dictionaries and text-books allot much space to it. English and American authors more correctly regard it as a symptomatic affection. Active fluxion to the lungs occurs with increased action of the heart, and when very hot air or irritating substances are inhaled. In diseases which interfere locally with the circulation the capillaries in the adjacent unaffected portions may be greatly distended. The importance, however, of this collateral fluxion, as it is called, is probably exaggerated. In a whole series of pulmonary affections there is this associated congestion—in pneumonia, bronchitis, pleurisy, and tuberculosis.

The symptoms of active congestion of the lungs are by no means definite. The description given by Woillez and by other French writers is of an affection which is difficult to recognize from anomalous or larval forms of pneumonia. The chief symptoms described are initial chill, pain in the side, dyspnœa, moderate cough, and temperature from 101° to 103°. The physical signs are defective resonance, feeble breathing, sometimes bronchial

in character, and fine rales. A majority of clinical physicians would undoubtedly class such cases under inflammation of the lung. In many epidemics the abnormal and larval forms are specially prevalent. This is no doubt the condition to which Porcher, of Charleston, called attention a short time ago as a "hitherto undescribed affection of the lungs."

The occurrence of an intense and rapidly fatal congestion of the lung, following extreme heat or cold or sometimes violent exertion, is recognized by some authors. Renforth, the oarsman, is said to have died from this cause during the race at Halifax. Leuf has described cases in which, in association with drunkenness, exposure, and cold, death occurred suddenly, or within twenty-four hours, the only lesion found being an extreme, almost hæmorrhagic, congestion of the lungs. It is by no means certain that in these cases death really occurs from pulmonary congestion in the absence of specific statements with reference to the coronary arteries. Several times in sudden death from disease of these vessels I have seen great engorgement of the lungs though not the extreme grade mentioned by Leuf. I have no personal knowledge of cases such as he describes.

(2) *Passive Congestion.*—Two forms of this may be recognized, the mechanical and the hypostatic.

(*a*) Mechanical congestion occurs whenever there is an obstacle to the return of the blood to the heart. It is a common event in many affections of the left heart. The lungs are voluminous, russet brown in color, cutting and tearing with great resistance. On section they show at first a brownish-red tinge, and then the cut surface, exposed to the air, becomes rapidly of a vivid red color from oxidation of the abundant hæmoglobin. This is the condition known as *brown induration* of the lung. Histologically it is characterized by (α) great distention of the alveolar capillaries; (β) increase in the connective-tissue elements of the lung; (γ) the presence in the alveolar walls of many cells containing altered blood-pigment; (δ) in the alveoli numerous epithelial cells containing blood-pigment in all stages of alteration, which are also found in great numbers in the sputum.

It occasionally happens that this mechanical hyperæmia of the lung results from pressure by tumors. So long as compensation is maintained the mechanical congestion of the lung in heart-disease does not produce any symptoms, but with enfeebled heart action the engorgement becomes marked and there are dyspnœa, cough, and expectoration, with the characteristic alveolar cells.

(*b*) Hypostatic congestion. In fevers and adynamic states generally, it is very common to find the bases of the lungs deeply congested, a condition induced partly by the effect of gravity, the patient lying recumbent in one posture for a long time, but chiefly by weakened heart action. That it is not an effect of gravity alone is shown by the fact that a healthy person may remain in bed an indefinite time without its occurrence. The term hypostatic congestion is applied to it. The posterior parts of the lung are dark in color and engorged with blood and serum; in some instances to such a degree that the alveoli no longer contain air and portions of the lung sink in water. The term *splenization* and hypostatic pneumonia have been given to these advanced grades. It is a common affection in protracted

cases of typhoid fever and in long debilitating illnesses. In ascites, meteorism, and abdominal tumors the bases of the lungs may be compressed and congested. In this connection must be mentioned the form of passive congestion met with in injury to, and organic disease of, the brain. In cerebral apoplexy the bases of the lungs are deeply engorged, not quite airless, but heavy, and on section drip with blood and serum. I have twice seen this condition in an extreme grade throughout the lungs in death from morphia poisoning. In some instances the lung tissue has a blackish, gelatinous, infiltrated appearance, almost like diffuse pulmonary apoplexy. Occasionally this congestion is most marked in, and even confined to, the hemiplegic side. In prolonged coma the hypostatic congestion may be associated with patches of consolidation, due to the aspiration of portions of food into the air-passages.

The symptoms of hypostatic congestion are not at all characteristic, and the condition has to be sought for by careful examination of the bases of the lungs, when slight dulness, feeble, sometimes blowing, breathing and liquid rales can be detected.

The *treatment* of congestion of the lungs is usually that of the condition with which it is associated. In the intense pulmonary engorgement, which may possibly occur primarily, and which is met with in heart-disease and emphysema, free bleeding should be practised. From 20 to 30 ounces of blood should be taken from the arm, and if the blood does not flow freely and the condition of the patient is desperate, aspiration of the right auricle may be performed.

Œdema.—In all forms of intense congestion of the lungs there is a transudation of serum from the engorged capillaries chiefly into the air-cells, but also into the alveolar walls. Not only is it very frequent in congestion, but also with inflammation, with new growths, infarcts, and tubercles. When limited to the neighborhood of an affected part, the name collateral œdema is sometimes applied to it. General œdema occurs under conditions very similar to those met with in congestion. It is very often, no doubt, a terminal event, occurring with the death agony. It is seen in typical form in the cachexias, in death from anæmia, also in chronic Bright's disease, disease of the heart, and cerebral affections.

The œdematous lung is heavy, looks watery, pits on pressure, and from the cut surface a large quantity of clear and, in cases of congestion, bloody serum flows freely; the tissue may even have a gelatinous, infiltrated appearance. The condition is much more common at the bases, but it may exist throughout the entire lung. The pathology of pulmonary œdema is not always clear. Two factors usually prevail in extreme cases—increased tension within the pulmonary system and a diluted blood plasma. The increased tension alone is not capable of producing it. The experiments of Welch seem to indicate that the essential factor lies in a disproportionate weakness of the left ventricle, so that the blood accumulates in the lung capillaries until transudation occurs, a view which satisfactorily explains certain cases, particularly the terminal œdemas.

The *symptoms* of œdema of the lungs are often only an aggravation of those already existing, and are due to the primary disease, whether car-

diac, renal, or general. There are usually increasing dyspnœa and cough, and on examination there may be defective resonance and large liquid rales at the bases. There are cases in which the œdema comes on with great suddenness, and in chronic Bright's disease it may prove rapidly fatal.

In the cases of so-called inflammatory œdema fever is always present, and there are often signs, more or less marked, of pneumonia.

The *treatment* of œdema of the lung is practically that of the conditions with which it is associated. In the acute cases active catharsis, and, if there is cyanosis, free venesection should be resorted to.

Pulmonary Hæmorrhage.—This occurs in two forms—*broncho-pulmonary hæmorrhage*, sometimes called bronchorrhagia, in which the blood is poured out into the bronchi and is expectorated, and *pulmonary apoplexy* or pneumorrhagia, in which the hæmorrhage takes place into the air-cells and the lung tissue.

1. *Broncho-pulmonary Hæmorrhage; Hæmoptysis.*—Spitting of blood, to which the term hæmoptysis should be restricted, results from a variety of conditions, among which the following are the most important: (*a*) In young healthy persons hæmoptysis may occur without warning, and after continuing for a few days disappear and leave no ill traces. There may be at the time of the attack no physical signs indicating pulmonary disease. In such cases good health may be preserved for years and no further trouble occur. These cases are not very uncommon. In Ware's important contribution to this subject,* of 386 cases of hæmoptysis noted in private practice 62 recovered and pulmonary disease did not subsequently develop in them. I know three professional men who had hæmoptysis as students, and who now, at periods of from fifteen to eighteen years subsequently, remain in perfect health. (*b*) Hæmoptysis in pulmonary tuberculosis, which is considered in pages 302–304. (*c*) In connection with certain diseases of the lung, as pneumonia (in the initial stage) and cancer, occasionally in gangrene, abscess, and bronchiectasis, hæmoptysis occurs. (*d*) Hæmoptysis is met with in many heart affections, particularly mitral lesions. It may be profuse and recur at intervals for years. (*e*) In ulcerative affections of the larynx, trachea, or bronchi. Sometimes the hæmorrhage is profuse and rapidly fatal, as when an ulcer erodes a large branch of the pulmonary artery, an accident which I have known to happen in a case of chronic bronchitis with emphysema. (*f*) Aneurism is an occasional cause of hæmoptysis. It may be sudden and rapidly fatal when the sac bursts into the air-passages. Slight bleeding may continue for weeks or even longer, due to pressure on the mucous membrane or erosion of the lung; or in some cases the sac "weeps" through the exposed laminæ of fibrin. (*g*) Vicarious hæmorrhage, which occurs in rare instances in cases of interrupted menstruation. The instances are well authenticated. Flint mentions a case which he had had under observation for four years, and Hippocrates refers to it in the aphorism, "Hæmoptysis in a woman is removed by an eruption of the menses." Periodical hæmoptysis has also been met with after the removal of both ovaries. Even fatal hæmorrhage has oc-

* On Hæmoptysis as a Symptom, by John Ware, M. D.

curred from the lung during menstruation when no lesion was found to acount for it. (*h*) There is a form of recurring hæmoptysis in arthritic subjects to which Sir Andrew Clark has called special attention and which also is described by French writers. The cases occur in persons over fifty years of age who usually present signs of the arthritic diathesis. It rarely leads to fatal issue and subsides without inducing pulmonary changes. (*i*) Hæmoptysis recurs sometimes in malignant fevers and in purpura hæmorrhagica. Lastly, there is endemic hæmoptysis, due to the *Distomum westermanni* in the bronchial tubes, an affection which is confined to parts of China and Japan.

Symptoms.—Hæmoptysis sets in as a rule suddenly. Often without warning the patient experiences a warm, saltish taste as the mouth fills with blood. Coughing is usually induced. There may be only an ounce or so brought up before the hæmorrhage stops, or the bleeding may continue for days, the patient bringing up small quantities. In other instances, particularly when a large vessel is eroded or an aneurism bursts, the amount is large, and the patient after a few attempts at coughing shows signs of suffocation and death is produced by inundation of the bronchial system. Fatal hæmorrhage may even occur into a large cavity in a patient debilitated by phthisis without the production of hæmoptysis. I dissected a case of this kind at the Philadelphia Hospital. The blood from the lungs generally has characters which render it readily distinguishable from the blood which is vomited. It is alkaline in reaction, frothy, and mixed with mucus, and when coagulation occurs air-bubbles are present in the clot. Blood-moulds of the smaller bronchi are sometimes seen. Patients can usually tell whether the blood has been brought up by coughing or by vomiting, and in a majority of cases the history gives important indications. In paroxysmal hæmoptysis connected with menstrual disturbances the practitioner should see that the blood is actually coughed up, since deception may be practised. The spurious hæmoptysis of hysteria is considered with that disease. Naturally, the patient is at first alarmed at the occurrence of bleeding, but, unless very profuse, as when due to rupture of an aortic aneurism in a pulmonary cavity, the danger is rarely immediate. The attacks, however, are apt to recur for a few days and the sputa may remain blood-tinged for a longer period. In the great majority of cases the hæmorrhage ceases spontaneously. It should be remembered that some of the blood may be swallowed and produce vomiting, and, after a day or two, the stools may be dark in color. It is not well during an attack of hæmoptysis to examine the chest. It was formerly thought that hæmorrhage exercised a prejudicial effect and excited inflammation of the lungs, but this is not often the case.

(2) *Pulmonary Apoplexy; Hæmorrhagic Infarct.*—In this condition the blood is effused into the air-cells and interstitial tissue. It is rarely indeed diffuse, the parenchyma being broken, as is the brain tissue in cerebral apoplexy. Sometimes, in disease of the brain, in septic conditions, and in the malignant forms of fevers, the lung tissue is uniformly infiltrated with blood and has, on section, a black, gelatinous appearance.

As a rule, the hæmorrhage is limited and results from the blocking of

The researches of Hoyden & Dixon indicate that all drugs which raise the peripheral blood pressure by vaso-constriction increase the clotting time of blood in the veins

a branch of the pulmonary artery either by a thrombus or an embolus. The condition is most common in chronic heart-disease. Although the pulmonary arteries are terminal ones, blocking is not always followed by infarction; partly because the wide capillaries furnish sufficient anastomosis, and partly because the bronchial vessels may keep up the circulation. The infarctions are chiefly at the periphery of the lung, usually wedge-shaped, with the base of the wedge toward the surface. When recent, they are dark in color, hard and firm, and look on section like an ordinary blood-clot. Gradual changes go on, and the color becomes a reddish brown. The pleura over an infarct is usually inflamed. A microscopical section shows the air-cells to be distended with red blood-corpuscles, which may also be in the alveolar walls. The infarcts are usually multiple and vary in size from a walnut to an orange. Very large ones may involve the greater part of a lobe. In the artery passing to the affected territory a thrombus or an embolus is found. The globular thrombi, formed in the right auricular appendix, play an important part in the production of hæmorrhagic infarction. In many cases the source of the embolus cannot be discovered, and the infarct may have resulted from thrombosis in the pulmonary artery, but, as before mentioned, it is not infrequent to find total obstruction of a large branch of a pulmonary artery without hæmorrhage into the corresponding lung area. The further history of an infarction is variable. It is possible that in some instances the circulation is re-established and the blood removed. More commonly, if the patient lives, the usual changes go on in the extravasated blood and ultimately a pigmented, puckered, fibroid patch results. Sloughing may occur with the formation of a cavity. Occasionally gangrene results. In a case at the University Hospital, Philadelphia, a gangrenous infarct ruptured and produced fatal pneumothorax.

The *symptoms* of pulmonary apoplexy are by no means definite. The condition may be suspected in chronic heart-disease when hæmoptysis occurs, particularly in mitral stenosis, but the bleeding may be due to the extreme engorgement. When the infarcts are very large, and particularly in the lower lobe, in which they most commonly occur, there may be signs of consolidation with blowing breathing.

Treatment of Pulmonary Hæmorrhage.—In the treatment of hæmoptysis it is important to remember the condition of the pulmonary circulation and the nature of the lesions associated with the hæmorrhage.

The pressure within the pulmonary artery is considerably less than that in the aortic system. We have as yet very imperfect knowledge of the circumstances which influence the lesser circulation in man. Researches, particularly those of Bradford, indicate that the system is under vasomotor control, but our knowledge of the mutual relations of pressure in the aorta and in the pulmonary artery, under varying conditions, is still very imperfect. Experiments with drugs seem to show that there may be an influence on systemic blood-pressure without any on the pulmonary, and the pressure in the one may rise while it falls in the other, or it may rise and fall in both together. In Andrew's Harveian Oration these rela-

tions are thoroughly described, and a statement is made, based on Bradford's experiments, as to the action on the pulmonary blood-pressure of many of the drugs employed in hæmoptysis. Thus ergot, the remedy perhaps most commonly used, causes a distinct rise in the pulmonary blood-pressure, while aconite produces a definite fall.

The anatomical condition in hæmoptysis is either hyperæmia of the bronchial mucosa (or of the lung tissue) or a perforated artery. In the latter case the patient often passes rapidly beyond treatment, though there are instances of the most profuse hæmorrhage, which must have come from a perforated artery or a ruptured aneurism, in which recovery has occurred. Practically, for treatment, we should separate these cases, as the remedies which would be applicable in a case of congested and bleeding mucosa would be as much out of place in a case of hæmorrhage from ruptured aneurism as in a cut radial artery. When the blood is brought up in large quantities, it is almost certain either that an aneurism has ruptured or a vessel has been eroded. In the instances in which the sputa are blood-tinged or when the blood is in smaller quantities, bleeding comes by diapedesis from hyperæmic vessels. In such cases the hæmorrhage may be beneficial in relieving the congested blood-vessels.

The indications are to reduce the frequency of the heart-beats and to lower the blood-pressure. By far the most important measure is absolute quiet of body, such as can only be secured by rest in bed and seclusion. In the majority of cases of mild hæmoptysis this is sufficient. Even when the patient insists upon going about, the bleeding may stop spontaneously. The diet should be light and unstimulating. Alcohol should not be used. The patient may, if he wishes, have ice to suck. Small doses of aromatic sulphuric acid may be given, but unless the bleeding is protracted styptic and astringent medicines are not indicated. For cough, which is always present and disturbing, opium should be freely given, and is of all medicines most serviceable in hæmoptysis. Digitalis should not be used, as it raises the blood-pressure in the pulmonary artery. Aconite, as it lowers the pressure, may be used when there is much vascular excitement. Ergot, tannic acid, and lead, which are so much employed, have little or no influence in hæmoptysis; ergot probably does harm. One of the most satisfactory means of lowering the blood-pressure is purgation, and when the bleeding is protracted salts may be freely given. In profuse hæmoptysis, such as comes from erosion of an artery or the rupture of an aneurism, a fatal result is common, and yet post-mortem evidence shows that thrombosis may occur with healing in a rupture of considerable size. The fainting induced by the loss of blood is probably the most efficient means of promoting thrombosis, and it was on this principle that formerly patients were bled from the arm, or from both arms, as in the case of Laurence Sterne. Ligatures, or Esmarch's bandages, placed around the legs may serve temporarily to check the bleeding. The ice-bag on the sternum is of doubtful utility. In a protracted case Cayley induced pneumothorax, but without effect.

Briefly, then, we may say that cases of hæmorrhage from rupture of aneurism or erosion of a blood-vessel usually prove fatal. The fainting

vessel.

Turn the patient on the affected side - gives
a the blood as the respiration is less apt to occur [illegible] the
bronchi of the sound lung. As oxaleus remarks in hemoptysis
the patient - dislikes for the first and needs to be strongly
reassured. Death rarely due directly to hemoptysis - patient
die after sink of [illegible] (Wood)

In ~~a few cases we have~~ used the hypo-
dermic injection of Salaine cc of a
1 or 2 % solution / I may be used of a prepare
to Ethanzley bleeding threatens + pure patient
saline ~~injection~~ / infusion.

induced by the loss of blood is beneficial, and, if the patient can be kept alive for twenty-four hours, a thrombus of sufficient strength to prevent further bleeding may form. The chief danger is the inundation of the bronchial system with the blood, so that while the hæmorrhage is profuse the cough should be encouraged. Opium should not then be used, and stimulants should be given with caution.

In the other group, in which the hæmorrhage comes from a congested area and is limited, the patient gets well if kept absolutely quiet, and fatal hæmorrhage probably never occurs from this source. Rest, reduction of the blood-pressure by minimum diet, purging, if necessary, and the administration of opium to allay the cough are the main indications.

II. BRONCHO-PNEUMONIA (*Capillary Bronchitis*).

This is essentially an inflammation of the terminal bronchus and the air-vesicles which make up a pulmonary lobule, whence the term broncho-pneumonia. It is also known as lobular, in contradistinction to lobar pneumonia. The term catarrhal is less applicable. The process begins usually with an inflammation of the capillary bronchi, which is a condition rarely, if ever, found without involvement of the lobular structures, so that it is now customary to consider the affections together. All forms of broncho-pneumonia depend upon invasion of the lung with microbes, and it would have been more consistent to place them with lobar pneumonia among the infectious disorders, but it is well perhaps to defer this until the bacteriology of the different varieties has been more fully worked out.

Etiology.—Broncho-pneumonia occurs either as a primary or as a secondary affection. The relative frequency in 443 cases is thus given by Holt: Primary, without previous bronchitis, 154; secondary (*a*) to bronchitis of larger tubes, 41; to measles, 89; to whooping-cough, 66; to diphtheria, 47; to scarlet fever, 7; to influenza, 6; to varicella, 2; to erysipelas, 2; and to acute ileo-colitis, 19. The proportion of primary to secondary forms as shown in this list is probably too low.

Primary acute broncho-pneumonia, like the lobar form, attacks children in good health, usually under two years. The etiological factors are very much those of ordinary pneumonia, and probably the pneumococcus is more often associated with it.

Secondary broncho-pneumonia occurs in two great groups: 1. As a sequence of the infectious fevers—measles, diphtheria, whooping-cough, scarlet fever, and, less frequently, small-pox, erysipelas, and typhoid fever. In children it forms the most serious complication of these diseases, and in reality causes more deaths than are due directly to the fevers. In large cities it ranks next in fatality to infantile diarrhœa. Following, as it does, the contagious diseases which principally affect children, we find that a large majority of cases occur during early life. According to Morrill's Boston statistics, it is most fatal during the first two years of life. The number of cases in a community increases or decreases with the prevalence of measles, scarlet fever, and diphtheria. It is most prevalent in the winter

and spring months. In the febrile affections of adults broncho-pneumonia is not very common. Thus in typhoid fever it is not so frequent as lobar pneumonia, though isolated areas of consolidation at the bases are by no means rare in protracted cases of this disease. In old people it is an extremely common affection, following debilitating causes of any sort, and supervening in the course of chronic Bright's disease and various acute and chronic maladies.

2. In the second division of this affection are embraced the cases of so-called aspiration or deglutition pneumonia. Whenever the sensitiveness of the larynx is benumbed, as in the coma of apoplexy or uræmia, minute particles of food or drink are allowed to pass the *rima*, and, reaching finally the smaller tubes, excite an intense inflammation similar to the vagus pneumonia which follows the section of the pneumogastrics in the dog. Cases are very common after operations about the mouth and nose, after tracheotomy, and in cancer of the larynx and œsophagus. The aspirated particles in some instances induce such an intense broncho-pneumonia that suppuration or even gangrene supervenes. The ether pneumonia, already described (p. 129), is often lobular in type.

An aspiration broncho-pneumonia may follow hæmoptysis (which has been already considered), the aspiration of material from a bronchiectatic cavity, and occasionally the material from an empyema which has ruptured into the lung.

A common and fatal form of broncho-pneumonia is that excited by the tubercle bacillus, which has already been considered.

Among general predisposing causes may be mentioned age. As just noted, it is prone to attack infants, and a majority of cases of pneumonia in children under five years of age are of this form. Of 370 cases in children under five years of age, 75 per cent were broncho-pneumonia (Holt). At the opposite extreme of life it is also common, in association with various debilitating circumstances and with the chronic diseases incident to the old. In children, rickets and diarrhœa are marked predisposing causes, and broncho-pneumonia is one of the most frequent post-mortem-room lesions in infants' homes and foundling asylums. The disease prevails most extensively among the poorer classes.

Morbid Anatomy.—On the pleural surfaces, particularly toward the base, are seen depressed bluish or blue-brown areas of collapse, between which the lung tissue is of a lighter color. Here and there are projecting portions over which the pleura may be slightly turbid or granular. The lung is fuller and firmer than normal, and, though in great part crepitant, there can be felt in places throughout the substance solid, nodular bodies. The dark depressed areas may be isolated or a large section of one lobe may be in the condition of collapse or atelectasis. Gradual inflation by a blowpipe inserted in the bronchus will distend a great majority of these collapsed areas. On section, the general surface has a dark reddish color and usually drips blood. Projecting above the level of the section are lighter red or reddish-gray areas representing the patches of broncho-pneumonia. These may be isolated and separated from each other by tracts of uninflamed tissue or they may be in groups; or the greater part of a lobe may

be involved. Study of a favorable section of an isolated patch shows: (*a*) A dilated central bronchiole full of tenacious purulent mucus. A fortunate section parallel to the long axis may show a racemose arrangement—the alveolar passages full of muco-pus. (*b*) Surrounding the bronchus for from 3 to 5 mm. or even more, an area of grayish-red consolidation, usually elevated above the surface and firm to the touch. Unlike the consolidation of lobar pneumonia, it may present a perfectly smooth surface, though in some instances it is distinctly granular. In a late stage of the disease small grayish-white points may be seen, which on pressure may be squeezed out as purulent droplets. A section in the axis of the lobule may present a somewhat grape-like arrangement, the stalks and stems representing the bronchioles and alveolar passages filled with a yellowish or grayish-white pus, while surrounding them is a reddish-brown hepatized tissue. (*c*) In the immediate neighborhood of this peribronchial inflammation the tissue is dark in color, smooth, airless, at a somewhat lower level than the hepatized portion, and differs distinctly in color and appearance from the other portions of the lung. This is the condition to which the term *splenization* has been given. It really represents a tissue in the early stage of inflammation, and it perhaps would be as well to give up the use of this term and also that of *carnification*, which is only a more advanced stage. The condition of collapse probably always precedes this, and it is difficult in some instances to tell the difference, as one shades into the other. In fact, collapse, splenization, and carnification are but preliminary steps in broncho-pneumonia.

While, in many cases, the areas of broncho-pneumonia present a reddish-brown color and are indistinctly granular, in others, particularly in adults, the nodules may resemble more closely gray hepatization and the air-cells are filled with a grayish, muco-purulent material. Minute hæmorrhages are sometimes seen in the neighborhood of the inflamed areas or on the pleural surfaces. Emphysema is commonly seen at the anterior borders and upper portions of the lung or in lobules adjacent to the inflamed ones. In many cases following diphtheria and measles the process is so extensive that the greater part of a lobe is involved, and it looks like a case of lobar hepatization. It has not, however, the uniformity of this affection, and collapsed dark strands may be seen between extensive areas of hepatized tissue.

There are three groups of cases: (1) Those in which the bronchitis and bronchiolitis are most marked, and in which there may be no definite consolidation, and yet on microscopical examination many of the alveolar passages and adjacent air-cells appear filled with inflammatory products. (2) The disseminated broncho-pneumonia, in which there are scattered areas of peribronchial hepatization with patches of collapse, while a considerable proportion of the lobe is still crepitant. This is by far the most common condition. (3) The pseudo-lobar form, in which the greater portion of the lobe is consolidated, but not uniformly, for intervening strands of dark congested lung tissue separate the groups of hepatized lobules.

Microscopically, the centre of the bronchus is seen filled with a plug of exudation, consisting of leucocytes and swollen epithelium. Section in

the long axis may show irregular dilatations of the tube. The bronchial wall is swollen and infiltrated with cells. Under a low power it is readily seen that the air-cells next the bronchus are most densely filled, while toward the periphery of the focus the alveolar exudation becomes less. The contents of the air-cells are made up of leucocytes and swollen endothelial cells in varying proportions. Red corpuscles are not often present and a fibrin network is rarely seen, though it may be present in some alveoli. In the swollen walls are seen distended capillaries and numerous leucocytes. As Delafield has pointed out, the interstitial inflammation of the bronchi and alveolar walls is the special feature of broncho-pneumonia.

The histological changes in the aspiration or deglutition broncho-pneumonia differ from the ordinary post-febrile form in a more intense infiltration of the air-cells with leucocytes, producing suppuration and foci of softening; even gangrene may be present.

Bacteriology of Broncho-pneumonia.—The organisms most commonly found in broncho-pneumonia are the *micrococcus lanceolatus*, the *streptococcus pyogenes* (either alone or with the pneumococcus), the *staphylococcus aureus et albus*, and Friedländer's *bacillus pneumoniæ*. The Klebs-Loeffler bacillus is not infrequently found in the secondary lesions of diphtheria. Except the pneumococcus these microbes are rarely found in pure cultures. In the lobular type the streptococcus is the most constant organism, in the pseudo-lobar the pneumococcus. Mixed infections are almost the rule in broncho-pneumonia.

M. Wollstein, in 17 primary cases, found the *micrococcus lanceolatus* alone in 9, with the streptococcus in 7. Of 14 secondary cases the *micrococcus lanceolatus* was found alone in 2 and with other organisms in 9. The primary form is the result of infection with the pneumococcus, the secondary most often with the streptococcus.

Terminations of Broncho-pneumonia.—(1) In *resolution*, which when it once begins goes on more rapidly than in fibrinous pneumonia. Broncho-pneumonia of the apices, in a child, persisting for three or more weeks, particularly if it follows measles or diphtheria, is often tuberculous. In these instances, when resolution is supposed to be delayed, caseation has in reality taken place. (2) In *suppuration*, which is rarely seen apart from the aspiration and deglutition forms, in which it is extremely common. (3) In *gangrene*, which occurs under the same conditions. (4) In *fibroid changes—chronic broncho-pneumonia*—a rare termination in the simple, a common sequence of the tuberculous, disease. Formerly it was thought that one of the most common changes in broncho-pneumonia, particularly in children, was caseation; but this is really a tuberculous process, the natural termination of an originally specific broncho-pneumonia. It is of course quite possible that a broncho-pneumonia, simple in its origin, may subsequently be the seat of infection by the *bacillus tuberculosis*.

Symptoms.—The *primary* form sets in abruptly with a chill or a convulsion. The child has not had a previous illness, but there may have been slight exposure. The temperature rises rapidly and is more constant; the physical signs are more local and there is not the widespread diffuse catarrh of the smaller tubes. Many cases are mistaken for lobar pneumonia. In

others the pulmonary features are in the background or are overlooked in the intensity of the general or cerebral symptoms. The termination is often by crisis, and the recovery is prompt. The mortality of this form is slight. S. West has recently (British Medical Journal, 1898, i) called attention to the importance of recognizing these primary cases and to their resemblance in clinical features with acute lobar pneumonia. The *secondary* form begins usually as a bronchitis of the smaller tubes. Much confusion has arisen from the description of capillary bronchitis as a separate affection, whereas it is only a part, though a primary and important one, of broncho-pneumonia. At the outset it may be said that if in convalescence from measles or in whooping-cough a child has an accession of fever with cough, rapid pulse, and rapid breathing, and if, on auscultation, fine rales are heard at the bases, or widely spread throughout the lungs, even though neither consolidation nor blowing breathing can be detected, the diagnosis of broncho-pneumonia may safely be made. I have never seen in a fatal case after diphtheria or measles a capillary bronchitis as the sole lesion. The onset is rarely sudden, or with a distinct chill; but after a day or so of indisposition the child gets feverish and begins to cough and to get short of breath. The fever is extremely variable; a range of from 102° to 104° is common. The skin is very dry and pungent. The cough is hard, distressing, and may be painful. Dyspnœa gradually becomes a prominent feature. Expiration may be jerky and grunting. The respirations may rise as high as 60 or even 80 per minute. Within the first forty-eight hours the percussion resonance is not impaired; the note, indeed, may be very full at the anterior borders of the lungs. On auscultation, many rales are heard, chiefly the fine subcrepitant variety, with sibilant rhonchi. There may really be no signs indicating that the parenchyma of the lung is involved, and yet even at this early stage, within forty-eight hours of the onset of the pulmonary symptoms, I have repeatedly, after diphtheria, found scattered nodules of lobular hepatization. Northrup, in a case in which death occurred within the first twenty-four hours, in addition to the extensive involvement of the smaller bronchi, found the intralobular tissue also involved in places. The dyspnœa is constant and progressive and soon signs of deficient aëration of the blood are noted. The face becomes a little suffused and the finger-tips bluish. The child has an anxious expression and gradually enters upon the most distressing stage of asphyxia. At first the urgency of the symptoms is marked, but soon the benumbing influence of the carbon dioxide on the nerve-centres is seen and the child no longer makes strenuous efforts to breathe. The cough subsides and, with a gradual increase in lividity and a drowsy restlessness, the right ventricle becomes more and more distended, the bronchial rales become more liquid as the tubes fill with mucus, and death occurs from heart paralysis. These are symptoms of a severe case of broncho-pneumonia, or what the older writers called *suffocative catarrh*.

The *physical signs* may at first be those of capillary bronchitis, as indicated by the absence of dulness, the presence of fine subcrepitant and whistling rales. In many cases death takes place before any definite pneumonic signs are detected. When these exist they are much more frequent

at the bases, where there may be areas of impaired resonance or even of positive dulness. When numerous foci involve the greater part of a lobe the breathing may become tubular, but in the scattered patches of ordinary broncho-pneumonia, following the fevers, the breathing is more commonly harsh than blowing. In grave cases there is retraction of the base of the sternum and of the lower costal cartilages during inspiration, pointing to deficient lung expansion.

Diagnosis.—With lobar pneumonia it may readily be confounded if the areas of consolidation are large and merged together. It is to be remembered, as Holt's figures well show, that broncho-pneumonia occurs chiefly in children under one year, whereas lobar pneumonia is more common after the third year. No writer has so clearly brought out the difference between pneumonia at these periods as Gerhard,* of Philadelphia, whose papers on this subject, though published nearly sixty years ago, have the freshness and accuracy which characterize all the writings of that eminent physician. Between lobar pneumonia and the secondary form of broncho-pneumonia the diagnosis is easy. The mode of onset is essentially different in the two infections, the one developing insidiously in the course or at the conclusion of another disease, the other setting in abruptly in a child in good health. In lobar pneumonia the disease is almost always unilateral, in broncho-pneumonia bilateral. The chief trouble arises in cases of primary broncho-pneumonia, which by aggregation of the foci involves the greater part of one lobe. Here the difficulty is very great, and the physical signs may be practically identical, but in broncho-pneumonia it is much more likely that a lesion, however slight, will be found on the other side.

A still more difficult question to decide is whether an existing broncho-pneumonia is simple or tuberculous. In many instances the decision cannot be made, as the circumstances under which the disease occurs, the mode of onset, and the physical signs may be identical. It has often been my experience that a case has been sent down from the children's ward to the dead-house with the diagnosis of post-febrile broncho-pneumonia in which there was no suspicion of the existence of tuberculosis; but on section there were found tuberculous bronchial glands and scattered areas of broncho-pneumonia, some of which were distinctly caseous, while others showed signs of softening. I have already spoken fully of this in the section on tuberculosis, but it is well to emphasize the fact that there are many cases of broncho-pneumonia in children which time alone enables us to distinguish from tuberculosis. The existence of extensive disease at the apices or central regions is a suggestive indication, and signs of softening may be detected. In the vomited matter, which is brought up after severe spells of coughing, sputum may be picked out and elastic tissue and bacilli detected.

It is a superfluous refinement to make a diagnosis between capillary bronchitis and catarrhal pneumonia, for the two conditions are part and parcel of the same disease. In simple bronchitis involving the larger tubes urgent dyspnœa and pulmonary distress are rarely present and the rales

* American Journal of the Medical Sciences, vols. xiv and xv.

are coarser and more sibilant. It must not be forgotten that, as in lobar pneumonia, cerebral symptoms may mask the true nature of the disease, and may even lead to the diagnosis of meningitis. I recall more than one instance in which it could not be satisfactorily determined whether the infant had tuberculous meningitis or a cerebral complication of an acute pulmonary affection.

Prognosis.—In the primary form the outlook is good. In children enfeebled by constitutional disease and prolonged fevers broncho-pneumonia is terribly fatal, but in cases coming on in connection with whooping-cough or after measles recovery may take place in the most desperate cases. It is in this disease that the truth of the old maxim is shown—"Never despair of a sick child." The death-rate in children under five has been variously estimated at from 30 to 50 per cent. After diphtheria and measles thin, wiry children seem to stand broncho-pneumonia much better than fat, flabby ones. In adults the aspiration or deglutition pneumonia is a very fatal disease.

Prophylaxis. Much can be done to reduce the probability of attack after febrile affections. Thus, in the convalescence from measles and whooping-cough, it is very important that the child should not be exposed to cold, particularly at night, when the temperature of the room naturally falls. In a nocturnal visit to the nursery—sometimes, too, I am sorry to say, to a children's hospital—how often one sees children almost naked, having kicked aside the bedclothes and having the night-clothes up about the arms! The use of light flannel "combinations" obviates this nocturnal chill, which is, I am sure, an important factor in the colds and pulmonary affections of young children, both in private houses and in institutions. The catarrhal troubles of the nose and throat should be carefully attended to, and during fevers the mouth should be washed two or three times a day with an antiseptic solution.

Treatment.—The frequency and the seriousness of broncho-pneumonia render it a disease which taxes to the utmost the resources of the practitioner. There is no acute pulmonary affection over which he at times so greatly despairs. On the other hand, there it not one in which he will be more gratified in saving cases which have seemed past all succor. The general arrangements should receive special attention. The room should be kept at an even temperature—about 65° to 68°—and the air should be kept moist with vapor.

At the outset the bowels should be opened by a mild purge, either castor oil or small doses of calomel, one twelfth to one sixth of a grain hourly until a movement is obtained, and care should be taken throughout the attack to secure a daily movement. The common saline fever mixture of citrate of potash, liquor ammonii acetatis, and aromatic spirits of ammonia may be given every two or three hours. If the disease comes on abruptly with high fever, minim or minim and a half doses of the tincture of aconite may be given with it. The pain, the distressing symptoms, and the incessant cough often demand opium, which must of course be used with care and judgment in the case of young children, but which is certainly not contra-indicated and may be usefully given in the form of

Dover's powder. Blisters are now rarely if ever employed, and even the jacket poultice has gone out of fashion. For the latter, however, I confess to a strong prejudice, and when lightly made and frequently changed it undoubtedly gives great relief. Much more commonly we now see, both in private and in hospital practice, the jacket of cotton-batting. Ice-poultices to the chest I have seen used apparently with great benefit, and they are warmly recommended by many German physicians as well as by Goodhart and others in England. The diet should consist of milk, broths, and egg albumen. Milk often curds and is disagreeable. Egg-white is particularly suitable and very acceptable when given in cold water with a little sugar. It forms, indeed, an excellent medium for the administration of the stimulants. If the pulse shows signs of failing, it is best to begin early with brandy. As in all febrile affections of children, cold water should be constantly at the bedside, and the child should be encouraged to drink freely. With these measures, in many cases the disease progresses to a favorable termination, but too often other and more serious symptoms arise. Cough becomes more distressing, dyspnœa increases, the ominous rattling of the mucus can be heard in the tubes, the child's color is not so good, and there is greater restlessness. Under these circumstances stimulant expectorants—ammonia, squills, and senega—should be given. Together they make a very disagreeable dose for a young child, particularly with the carbonate of ammonia. The aromatic spirits of ammonia is somewhat better. If the carbonate is employed, it must be given in small doses, not more than a grain to an infant of eighteen months. If the child has increasing difficulty in getting up the mucus, an emetic should be given—either the wine of ipecac or, if necessary, tartar emetic. There is no necessity, however, to keep the child constantly nauseated. Enough should be given to cause prompt emesis, and the benefit results in the expulsion of mucus from the larger tubes. In this stage, too, strychnine is undoubtedly helpful in stimulating the depressed respiratory centre. With commencing cyanosis, inhalations of oxygen may be employed, sometimes with great benefit.

With rapid failure of the heart, loud mucous rattles in the throat, and increasing lividity, every measure should be used to arouse the child and excite coughing. Alternate douches of hot and cold water, electricity, which I have seen applied with good results at Wiederhofer's clinic in Vienna, and hypodermic injections of ether may be tried. For the reduction of temperature, particularly if cerebral symptoms are prominent, there is nothing so satisfactory as the wet pack or the cold bath. In the case of children, when the latter is used it should be graduated, beginning with a temperature which is pleasantly warm and gradually reducing it to 75° or 80°. Even when the temperature is not high, the cerebral symptoms are greatly relieved by the bath or the pack.

III. CHRONIC INTERSTITIAL PNEUMONIA

(*Cirrhosis of the Lung—Fibroid Phthisis*).

This consists in the gradual substitution to a greater or less extent of connective tissue for the normal lung. It is a fibroid change which may have its starting-point in the tissue about the bronchi and blood-vessels, the interlobular septa, the alveolar walls, or in the pleura. So diverse are the different forms and so varied the conditions under which this change occurs that a proper classification is extremely difficult. We may recognize, however, two chief forms—the *local*, which involves only a limited area of the lung substance, and the *diffuse*, invading either both lungs or an entire organ.

Etiology.—*Local* fibroid change in the lungs is common. It is a constant accompaniment of tubercle and in every case of phthisis the chronic interstitial changes play a very important *rôle*. In tumors, abscess, gummata, hydatids, and emphysema it also occurs. Fibroid processes are frequently met with at the apices of the lung and may be due either to a limited healed tuberculosis, to fibroid induration in consequence of pigment, or, in a few instances, may result from thickening of the pleura. They have been described at page 331.

Diffuse interstitial pneumonia is met with under the following circumstances: 1. As a sequence of acute fibrinous pneumonia. Although extremely rare, this is recognized as a possible termination. From unknown causes resolution fails to take place. A gradual process of organization goes on in the fibrinous plugs within the air-cells and the alveolar walls become greatly thickened by a new growth, first of nuclear and subsequently of fibrillated connective tissue. Macroscopically there is produced a smooth, grayish, homogeneous tissue which has the peculiar translucency of all new-formed connective tissue. This has been called gray induration. A majority of the cases terminate within a few months, and instances which have been followed from the outset are very rare.

2. *Chronic Broncho-Pneumonia.*—The relation of broncho-pneumonia to cirrhosis of the lung has been specially studied by Charcot, who states that it may follow the acute or subacute form of this disease, particularly in children. The fibrosis extends from the bronchi, which are usually found dilated. Bronchiectasis itself may be followed by fibrosis of the lung. The alveolar walls are thickened and the lobules converted into firm grayish masses, in which there is no trace of normal lung tissue. This process may go on and involve an entire lobe or even the whole lung. Many of these cases are tuberculous from the outset.

3. *Pleurogenous Interstitial Pneumonia.*—Charcot applies this term to that form of cirrhosis of the lung which follows invasion from the pleura. Doubt has been expressed by some writers whether this really occurs. While Wilson Fox is probably correct in questioning whether an entire lung can become cirrhosed by the gradual invasion from the pleura, there can be no doubt that there are instances of primitive dry pleurisy, which,

as Sir Andrew Clark has pointed out, gradually compresses the lung and at the same time leads to interstitial cirrhosis. This may be due in part to the fibroid change which follows prolonged compression. In some cases there seems to be a distinct connection between the greatly thickened pleura and the dense strands of fibrous tissue passing from it into the lung substance. Instances occur in which one lobe or the greater part of it presents, on section, a mottled appearance, owing to the increased thickness of the interlobar septa—a condition which may exist without a trace of involvement of the pleura. In many other cases, however, the extension seems to be so definitely associated with pleurisy that there is no doubt as to the causal connection between the two processes. In these instances the lung is removed with great difficulty, owing to the thickness and close adhesion of the pleura to the chest wall.

4. *Chronic interstitial pneumonia*, due to inhalation of dust, which is considered in a separate section.

5. *Syphilis* of the lung presents the features of a chronic fibrosis of the organ (see p. 247).

6. Indurative changes in the lung may follow the compression by aneurism or new growth or the irritation of a foreign body in a bronchus.

Morbid Anatomy.—There are two chief forms, the massive or lobar and the insular or broncho-pneumonic form. In the massive type the disease is unilateral; the chest of the affected side is sunken, deformed, and the shoulder much depressed. On opening the thorax the heart is seen drawn far over to the affected side. The unaffected lung is emphysematous and covers the greater portion of the mediastinum. It is scarcely credible in how small a space, close to the spine, the cirrhosed lung may lie. The adhesions between the pleural membranes may be extremely dense and thick, particularly in the pleurogenous cases; but when the disease has originated in the lung there may be little thickening of the pleura. The organ is airless, firm, and hard. It strongly resists cutting, and on section shows a grayish fibroid tissue of variable amount, through which pass the blood-vessels and bronchi. The latter may be either slightly or enormously dilated. There are instances in which the entire lung is converted into a series of bronchiectatic cavities and the cirrhosis is apparent only in certain areas or at the root. The tuberculous cases can usually be differentiated by the presence of an apical cavity, not bronchiectatic, and often large; and the other lung almost invariably shows tuberculous lesions. Pulmonary aneurisms are not infrequent in the cavities. The other lung is always greatly enlarged and emphysematous. The heart is hypertrophied, particularly the right ventricle, and there may be marked atheromatous changes in the pulmonary artery. An amyloid condition of the viscera is found in some cases.

In the broncho-pneumonic form the areas are smaller, often centrally placed, and most frequently in the lower lobes. They are deeply pigmented, show dilated bronchi, and when multiple are separated by emphysematous lung tissue.

A *reticular form* of fibrosis of the lung has been described by Percy

Kidd and W. McCollum, in which the lungs are intersected by grayish fibroid strands following the lines of the interlobular septa.

Symptoms and Course.—The disease is essentially chronic, extending over a period of many years, and when once the condition is established the health may be fairly good. In a well-marked case the patient complains only of his chronic cough, perhaps a slight shortness of breath. In other respects he is quite well, and is usually able to do light work. The cases are commonly regarded as phthisical, though there may be scarcely a symptom of that affection except the cough. There are instances, however, of fibroid phthisis which cannot be distinguished from cirrhosis of the lung except by the presence of tubercle bacilli in the expectoration. As the bronchi are usually dilated, the symptoms and physical signs may be those of bronchiectasis. The cough is paroxysmal and the expectoration is generally copious and of a muco-purulent or sero-purulent nature. It is sometimes fetid. Hæmorrhage is by no means infrequent, and occurred in more than one half of the cases analyzed by Bastian. Walking on the level and in the ordinary affairs of life the patient may show no shortness of breath, but in the ascent of stairs and on exertion there may be dyspnœa.

Physical Signs.—*Inspection.*—The affected side is immobile, retracted, and shrunken, and contrasts in a striking way with the voluminous sound side. The intercostal spaces are obliterated and the ribs may even overlap. The shoulder is drawn down and from behind it is seen that the spine is bowed. The heart is greatly displaced, being drawn over by the shrinkage of the lung to the affected side. When the left lung is affected there may be a large area of visible impulse in the second, third, and fourth interspaces. Mensuration shows a great diminution in the affected side, and with the saddle-tape the expansion may be seen to be negative. The *percussion* note varies with the condition of the bronchi. It may be absolutely flat, particularly at the base or at the apex. In the axilla there may be a flat tympany or even an amphoric note over a large sacculated bronchus. On the opposite side the percussion note is usually hyperresonant. On *auscultation* the breath-sounds have either a cavernous or amphoric quality at the apex, and at the base are feeble, with mucous, bubbling rales. The voice-sounds are usually exaggerated. Cardiac murmurs are not uncommon, particularly late in the disease, when the right heart fails. These are, of course, the physical signs of the disease when it is well established. They naturally vary considerably, according to the stage of the process. The disease is essentially chronic, and may persist for fifteen or twenty years. Death occurs sometimes from hæmorrhage, more commonly from gradual failure of the right heart with dropsy, and occasionally from amyloid degeneration of the organs.

The *diagnosis* is never difficult. It may be impossible to say, without a clear history, whether the origin is pleuritic or pneumonic. Between cases of this kind and fibroid phthisis it is not always easy to discriminate, as the conditions may be almost identical. When tuberculosis is present, however, even in long-standing cases, bacilli are usually present in the sputa, and there may be signs of disease in the other lung.

Treatment.—It is only for an intercurrent affection or for an aggravation of the cough that the patient seeks relief. Nothing can be done for the condition itself. When possible the patient should live in a mild climate, and should avoid exposure to cold and damp. A distressing feature in some cases is the putrefaction of the contents of the dilated tubes, for which the same measures may be used as in fetid bronchitis.

IV. PNEUMONOKONIOSIS.

Under this term, introduced by Zenker, are embraced those forms of fibrosis of the lung due to the inhalation of dusts in various occupations. They have received various names, according to the nature of the inhaled particles—*anthracosis,* or coal-miner's disease; *siderosis,* due to the inhalation of metallic dusts, particularly iron; *chalicosis,* due to the inhalation of mineral dusts, producing the so-called stone-cutter's phthisis, or the "grinder's rot" of the Sheffield workers.

The dust particles inhaled into the lungs are dealt with extensively by the ciliated epithelium and by the phagocytes, which exist normally in the respiratory organs. The ordinary mucous corpuscles take in a large number of the particles, which fall upon the trachea and main bronchi. The cilia sweep the mucus out to a point from which it can be expelled by coughing. It is doubtful if the particles ever reach the air-cells, but the swollen alveolar cells (in which they are in numbers) probably pick them up on the way. The mucous and the alveolar cells are the normal respiratory scavengers. In dwellers in the country, in which the air is pure, they are able to prevent the access of dust particles to the lung tissue, so that even in adults these organs present a rosy tint, very different from the dark, carbonized appearance of the lungs of dwellers in cities. When the impurities in the air are very abundant, a certain proportion of the dust particles escapes these cells and penetrates the mucosa, reaching the lymph spaces, where they are attacked at once by the cells of the connective-tissue stroma, which are capable of ingesting and retaining a large quantity. In coal-miners, coal-heavers, and others whose occupations necessitate the constant breathing of a very dusty atmosphere even these forces are insufficient. Many of the particles enter the lymph stream and, as Arnold has shown in his beautiful researches, are carried (1) to the lymph nodules surrounding the bronchi and blood-vessels; (2) to the interlobular septa beneath the pleura, where they lodge in and between the tissue elements; and (3) along the larger lymph channels to the substernal, bronchial and tracheal glands, in which the stroma cells of the follicular cords dispose of them permanently and prevent them from entering the general circulation. Occasionally in anthracosis the carbon grains do reach the general circulation, and the coal dust is found in the liver and spleen. As Weigert has shown, this occurs when the densely pigmented bronchial glands closely adhere to the pulmonary veins, through the walls of which the carbon particles pass to the general circulation. The lung tissue has a remarkable tolerance for these particles, probably because a large propor-

tion of them is warehoused, so to speak, in protoplasmic cells. By constant exposure a limit is reached, and there is brought about a very definite pathological condition, an interstitial sclerosis. In coal-miners this may occur in patches, even before the lung tissue is uniformly infiltrated with the dust. In others it appears only after the entire organs have become so laden that they are dark in color, and an ink-like juice flows from the cut surface. The lungs of a miner may be black throughout and yet show no local lesions and be everywhere crepitant.

As already mentioned, the particles are deposited in large numbers in the follicular cords of the tracheal and bronchial glands and of the peri-bronchial and peri-arterial lymph nodules, and in these they finally excite proliferation of the connective-tissue elements. It is by no means uncommon to find in persons whose lungs are only moderately carbonized the bronchial glands sclerosed and hard. In anthracosis the fibroid changes usually begin in the peri-bronchial lymph tissue, and in the early stage of the process the sclerosis may be largely confined to these regions. A Nova Scotian miner, aged thirty-six, died under my care, at the Montreal General Hospital, of black small-pox, after an illness of a few days. In his lungs (externally coal-black) there were round and linear patches ranging in size from a pea to a hazel-nut, of an intensely black color, airless and firm, and surrounded by a crepitant tissue, slate-gray in color. In the centre of each of these areas was a small bronchus. Many of them were situated just beneath the pleura, and formed typical examples of limited fibroid broncho-pneumonia. In addition there is usually thickening of the alveolar walls, particularly in certain areas. By the gradual coalescence of these fibroid patches large portions of the lung may be converted into firm grayish-black, in the case of the coal-miner—steel-gray, in the case of the stone-worker—areas of cirrhosis. In the case of a Cornish miner, aged sixty-three, who died under my care, one of these fibroid areas measured 18 by 6 cm. and 4.5 cm. in depth.

A second important factor in these cases is chronic bronchitis, which is present in a large proportion and really causes the chief symptoms. A third is the occurrence of emphysema, which is almost invariably associated with long-standing cases of pneumonokoniosis. With the changes so far described, unless the cirrhotic area is unusually extensive, the case may present the features of chronic bronchitis with emphysema, but finally another element comes into play. In the fibroid areas softening occurs, probably a process of necrosis similar to that by which softening is produced in fibro-myomata of the uterus. At first these are small and contain a dark liquid. Charcot calls them *ulcères du poumon*. They rarely attain a large size unless a communication is formed with the bronchus, in which case they may become converted into suppurating cavities. The question has been much discussed of late as to what part the tubercle bacillus plays in these cases of pneumonokoniosis with cavity formation. In some instances there is certainly a tuberculous process ingrafted, but that large excavations may occur, or in other instances bronchiectasis without the presence of bacilli, I have convinced myself by the examination of several characteristic specimens.

The *siderosis* induced by the oxide of iron causes an interstitial pneumonia similar to anthracosis. Workers in brass and in bronze are liable to a like affection.

Chalicosis, due to the deposit of particles of silex and alumina, is found in the makers of mill-stones, particularly the French mill-stones, and also in knife and axe grinders and stone-cutters. Anatomically, this form is characterized by the production of nodules of various sizes, which are cut with the greatest difficulty and sometimes present a curious grayish, even glittering, crystalloid appearance.

Workers in flax and in cotton, and grain-shovellers are also subject to these chronic interstitial changes in the lungs. In all these occupations, as shown by Greenhow, to whose careful studies we owe so much of our knowledge of these diseases, the condition of the lung may ultimately be almost identical.

The *symptoms* do not come on until the patient has worked for a variable number of years in the dusty atmosphere. As a rule there are cough and failing health for a prolonged period of time before complete disability. The coincident emphysema is responsible in great part for the shortness of breath and wheezy condition of these patients. The expectoration is usually muco-purulent, often profuse; in a case of anthracosis, very dark in color—the so-called "black spit"; in a case of chalicosis there may be seen under the microscope the bright angular particles of silica.

Even when there are physical signs of cavity, tubercle bacillus are not necessarily, and indeed in my experience they are not usually present. It is remarkable for how long a time a coal-miner may continue to bring up sputum laden with coal particles even when there are only signs of a chronic bronchitis. Many of the particles are contained in the cells of the alveolar epithelium. In these instances it appears that an attempt is made by the leucocytes to rid the lungs of some of the carbon grains.

The *diagnosis* of the condition is rarely difficult; the expectoration is usually characteristic. It must always be borne in mind that chronic bronchitis and emphysema form essential parts of the process and that in late stages there may be tuberculous infection.

The *treatment* of the condition is practically that of chronic bronchitis and emphysema.

V. EMPHYSEMA.

Definition.—The condition in which the infundibular passages and the alveoli are dilated and the alveolar walls atrophied.

A practical division may be made into compensatory, hypertrophic, and atrophic forms, the acute vesicular emphysema, and the interstitial forms. The last two do not in reality come under the above definition, but for convenience they may be considered here.

I. Compensatory Emphysema.

Whenever a region of the lung does not expand fully in inspiration, either another portion of the lung must expand or the chest wall sink in order to occupy the space. The former almost invariably occurs. We have already mentioned that in broncho-pneumonia there is a vicarious distention of the air-vesicles in the adjacent healthy lobules, and the same happens in the neighborhood of tuberculous areas and cicatrices. In general pleural adhesions there is often compensatory emphysema, particularly at the anterior margins of the lung. The most advanced example of this form is seen in cirrhosis, when the unaffected lung increases greatly in size, owing to distention of the air-vesicles. A similar though less marked condition is seen in extensive pleurisy with effusion and in pneumothorax.

At first, this distention of the air-vesicles is a simple physiological process and the alveolar walls are stretched but not atrophied. Ultimately, however, in many cases they waste and the contiguous air-cells fuse, producing true emphysema.

II. Hypertrophic Emphysema.

The large-lunged emphysema of Jenner, also known as substantive or idiopathic emphysema, is a well-marked clinical affection, characterized by enlargement of the lungs, due to distention of the air-cells and atrophy of their walls, and clinically by imperfect aëration of the blood and more or less marked dyspnœa.

Etiology.—Emphysema is the result of persistently high intra-alveolar tension acting upon a congenitally weak lung tissue. If the mechanical views as to its origin, which have prevailed so long, were true, the disease would certainly be much more common; since violent respiratory efforts, believed to be the essential factor, are performed by a majority of the working classes. Strongly in favor of the view, that the nutritive change in the air-cells is the primary factor, is the markedly hereditary character of the disease and the frequency with which it starts early in life. These are two points upon which scarcely sufficient stress has been laid. To James Jackson, Jr., of Boston, we owe the first observations on the hereditary character of emphysema. Working under Louis' directions, he found that in 18 out of 28 cases one or both parents were affected.

I have been impressed by the frequency of its origin in childhood. It may follow recurring asthmatic attacks due to adenoid vegetations. It may develop, too, in several members of the same family. We are still ignorant as to the nature of this congenital pulmonary weakness. Cohnheim thinks it probably due to a defect in the development of the elastic-tissue fibres—a statement which is borne out by Eppinger's observations.

Heightened pressure within the air-cells may be due to forcible inspiration or expiration. Much discussion has taken place as to the part played by these two acts in the production of the disease. The inspiratory

theory was advanced by Laennec and subsequently modified by Gairdner, who held that in chronic bronchitis areas of collapse were induced, and compensatory distention took place in the adjacent lobules. This unquestionably does occur in the vicarious or compensatory emphysema, but it probably is not a factor of much moment in the form now under consideration. The expiratory theory, which was supported by Mendelssohn and Jenner, accounts for the condition in a much more satisfactory way. In all straining efforts and violent attacks of coughing, the glottis is closed and the chest walls are strongly compressed by muscular efforts, so that the strain is thrown upon those parts of the lung least protected, as the apices and the anterior margins, in which we always find the emphysema most advanced. The sternum and costal cartilages gradually yield to the heightened intrathoracic pressure and are, in advanced cases, pushed forward, giving the characteristic rotundity to the thorax. The cartilages gradually become calcified. One theory of the disease is that there is a gradual enlargement of the thorax and the lungs increase in volume to fill up the space.

Of other etiological factors occupation is the most important. The disease is met with in players on wind instruments, in glass-blowers, and in occupations necessitating heavy lifting or straining. Whooping-cough and bronchitis play an important *rôle*, not so much in the changes which they induce in the bronchi as in consequence of the prolonged attacks of coughing.

Morbid Anatomy.—The thorax is capacious, usually barrel-shaped, and the cartilages are calcified. On removal of the sternum, the anterior mediastinum is found completely occupied by the edges of the lungs, and the pericardial sac may not be visible. The organs are very large and have lost their elasticity, so that they do not collapse either in the thorax or when placed on the table. The pleura is pale and there is often an absence of pigment, sometimes in patches, termed by Virchow *albinism* of the lung. To the touch they have a peculiar, downy, feathery feel, and pit readily on pressure. This is one of the most marked features. Beneath the pleura greatly enlarged air-vesicles may be readily seen. They vary in size from $\frac{1}{2}$ to 3 mm., and irregular bullæ, the size of a walnut or larger, may project from the free margins. The best idea of the extreme rarefaction of the tissue is obtained from sections of a lung distended and dried. At the anterior margins the structure may form an irregular series of air-chambers, resembling the frog's lung. On careful inspection with the hand-lens, remnants of the interlobular septa or even of the alveoli may be seen on these large emphysematous vesicles. Though general throughout the organs, the distention is more marked, as a rule, at the anterior margins, and is often specially developed at the inner surface of the lobe near the root, where in extreme cases air-spaces as large as an egg may sometimes be found. Microscopically there is seen atrophy of the alveolar walls, by which is produced the coalescence of neighboring air-cells. In this process the capillary network disappears before the walls are completely atrophied. The loss of the elastic tissue is a special feature. It is stated, indeed, that in certain cases there is a congenital

defect in the development of this tissue. The epithelium of the air-cells undergoes a fatty change, but the large distended air-spaces retain a pavement layer.

The bronchi show important changes. In the larger tubes the mucous membrane may be rough and thickened from chronic bronchitis; often the longitudinal lines of submucous elastic tissue stand out prominently. In the advanced cases many of the smaller tubes are dilated, particularly when, in addition to emphysema, there are peri-bronchial fibroid changes. Bronchiectasis is not, however, an invariable accompaniment of emphysema, but, as Laennec remarks, it is difficult to understand why it is not more common. Of associated morbid changes the most important are found in the heart. The right chambers are dilated and hypertrophied, the tricuspid orifice is large, and the valve segments are often thickened at the edges. In advanced cases the cardiac hypertrophy is general. The pulmonary artery and its branches may be wide and show marked atheromatous changes.

The changes in the other organs are those commonly associated with prolonged venous congestion.

Symptoms.—The disease may be tolerably advanced before any special symptoms develop. A child, for instance, may be somewhat short of breath on going up-stairs or may be unable to run and play as other children without great discomfort; or, perhaps, has attacks of slight lividity. Doubtless much depends upon the completeness of cardiac compensation. When this is perfect, there may be no special interruption of the pulmonary circulation and, except with violent exertion, there is no interference with the aëration of the blood. In well-developed cases the following are the most important symptoms: *Dyspnœa*, which may be felt only on slight exertion, or may be persistent, and aggravated by intercurrent attacks of bronchitis. The respirations are often harsh and wheezy, and expiration is distinctly prolonged.

Cyanosis of an extreme grade is more common in emphysema than in other affections with the exception of congenital heart-disease. So far as I know it is the only disease in which a patient may be able to go about and even to walk into the hospital or consulting-room with a lividity of startling intensity. The contrast between the extreme cyanosis and the comparative comfort of the patient is very striking. In other affections of the heart and lungs associated with a similar degree of cyanosis the patient is invariably in bed and usually in a state of orthopnœa. One condition must be here referred to, viz., the extraordinary cyanosis in cases of poisoning by aniline products, which is in most part due to the conversion of the hæmoglobin into methæmoglobin.

Bronchitis with associated cough is a frequent symptom and often the direct cause of the pulmonary distress. The contrast between emphysematous patients in the winter and summer is marked in this respect. In the latter they may be comfortable and able to attend to their work, but with the cold and changeable weather they are laid up with attacks of bronchitis. Finally, in fact, the two conditions become inseparable and the patient has persistently more or less cough. The acute

bronchitis may produce attacks not unlike asthma. In some instances this is true spasmodic asthma, with which emphysema is frequently associated.

As age advances, and with successive attacks of bronchitis, the condition gets slowly worse. In hospital practice it is common to admit patients over sixty with well-marked signs of advanced emphysema. The affection can generally be told at a glance—the rounded shoulders, barrel chest, the thin yet oftentimes muscular form, and sometimes, I think, a very characteristic facial expression.

There is another group, however, of younger patients from twenty-five to forty years of age who, winter after winter, have attacks of intense cyanosis in consequence of an aggravated bronchial catarrh. On inquiry we find that these patients have been short-breathed from infancy, and they belong, I believe, to a category in which there has been a primary defect of structure in the lung tissue.

Physical Signs.—*Inspection.*—The thorax is markedly altered in shape; the antero-posterior diameter is increased and may be even greater than the lateral, so that the chest is barrel-shaped. The appearance is somewhat as if the chest was in a permanent inspiratory position. The sternum and costal cartilages are prominent. The lower zone of the thorax looks large and the intercostal spaces are much widened, particularly in the hypochondriac regions. The sternal fossa is deep, the clavicles stand out with great prominence, and the neck looks shortened from the elevation of the thorax and the sternum. A zone of dilated venules may be seen along the line of attachment of the diaphragm. Though this is common in emphysema, it is by no means peculiar to it or indeed to any special affection. Andrew, of Bartholomew's Hospital, and, according to Duckworth, Laycock called attention to it.

The curve of the spine is increased and the back is remarkably rounded, so that the scapulæ seem to be almost horizontal. Mensuration shows the rounded form of the chest and the very slight expansion on deep inspiration. The respiratory movements, which may look energetic and forcible, exercise little or no influence. The chest does not expand, but there is a general elevation. The inspiratory effort is short and quick; the expiratory movement is prolonged. There may be retraction instead of distention in the upper abdominal region during inspiration, and there is sometimes seen a transverse curve crossing the abdomen at the level of the twelfth rib. The apex beat of the heart is not visible, and there is usually marked pulsation in the epigastric region. The cervical veins stand out prominently and may pulsate.

Palpation.—The vocal fremitus is somewhat enfeebled but not lost. The apex beat can rarely be felt. There is a marked shock in the lower sternal region and very distinct pulsation in the epigastrium. *Percussion* gives greatly increased resonance, full and drum-like—what is sometimes called hyperresonance. The note is not often distinctly tympanitic in quality. The percussion note is greatly extended, the heart dulness may be obliterated, the upper limit of liver dulness is greatly lowered, and the resonance may extend to the costal margin. Behind, a clear percussion note

extends to a much lower level than normal. The level of splenic dulness, too, may be lowered.

On *auscultation* the breath-sounds are usually enfeebled and may be masked by bronchitic rales. The most characteristic feature is the prolongation of the expiration, and the normal ratio may be reversed—4 to 1 instead of 1 to 4. It is often wheezy and harsh and associated with coarse rales and sibilant rhonchi. It is said that in interstitial emphysema there may be a friction sound heard, not unlike that of pleurisy. The heart-sounds are usually clear; but in advanced cases, when there is marked cyanosis, a tricuspid regurgitant murmur may be heard. Accentuation of the pulmonary second sound is present.

The *course* of the disease is slow but progressive, the recurring attacks of bronchitis aggravating the condition. Death may occur from intercurrent pneumonia, either lobar or lobular, and dropsy may supervene from cardiac failure. Occasionally death results from overdistention of the heart, with extreme cyanosis. Duckworth has called attention to the occasional occurrence of fatal hæmorrhage in emphysema. In an old emphysematous patient at the Montreal General Hospital death followed the erosion of a main branch of the pulmonary artery by an ulcer near the bifurcation of the trachea.

Treatment.—Practically, the measures mentioned in connection with bronchitis should be employed. In children with asthma and developing emphysema the nose should be carefully examined. No remedy is known which has any influence over the progress of the condition itself. Bronchitis is the great danger of these patients, and therefore when possible they should live in an equable climate. In consequence of the venous engorgement they are liable to gastric and intestinal disturbance, and it is particularly important to keep the bowels regulated and to avoid flatulency which often seriously aggravates the dyspnœa. Patients who come into the hospital in a state of urgent dyspnœa and lividity, with great engorgement of the veins, particularly if they are young and vigorous, should be bled freely. On more than one occasion I have saved the lives of persons in this condition by venesection. Inhalation of oxygen may be used and the remedies given already mentioned in connection with bronchitis. Strychnine will be found specially useful.

III. Atrophic Emphysema.

This is really a senile change and is called by Sir William Jenner small-lunged emphysema. It is really a primary atrophy of the lung, coming on in advanced life, and scarcely constitutes a special affection. It occurs in "withered-looking old persons" who may perhaps have had a winter cough and shortness of breath for years. In striking contrast to the essential or hypertrophic emphysema, the chest in this form is small. The ribs are obliquely placed, the decrease in the diameter being due to greatly increased obliquity in the position of the ribs. The thoracic muscles are usually atrophied. In advanced cases of this affection the lung presents a remarkable appearance, being converted into a series of large vesicles, on

the walls of which the remnants of air-cells may be seen. It is a condition for which nothing can be done.

IV. Acute Vesicular Emphysema.

When death occurs from bronchitis of the smaller tubes, or from cyanosis when strong inspiratory efforts have been made, the lungs are large in volume and the air-cells are much distended. Clinically, this condition may develop rapidly in cases of cardiac asthma and angina pectoris. The lungs are voluminous, the area of pulmonary resonance is much increased, and on auscultation there are heard everywhere piping rales and prolonged expiration. It is the condition to which von Basch has given the names *Lungenschwellung* and *Lungenstarrheit*. A similar condition may follow pressure on the vagi.

V. Interstitial Emphysema.

In this form beads of air are seen in the interlobular and subpleural tissue; sometimes they form large bullæ beneath the pleura. A rare event is rupture close to the root of the lung, and the passage of air along the trachea into the subcutaneous tissues of the neck. After tracheotomy just the reverse may occur and the air may pass from the tracheotomy wound along the wind-pipe and bronchi and appear beneath the surface of the pleura. From this interstitial emphysema spontaneous pneumothorax may arise in healthy persons.

VI. GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is not an affection *per se*, but occurs in a variety of conditions when necrotic areas undergo putrefaction. It it not easy to say why sphacelus should occur in one case and not in another, as the germs of putrefaction are always in the air-passages, and yet necrotic territories rarely become gangrenous. Total obstruction of a pulmonary artery, as a rule, causes infarction, and the area shut off does not often, though it may, sphacelate. Another factor would seem to be necessary—probably a lowered tissue resistance, the result of general or local causes. It is met with (1) as a sequence of lobar pneumonia. This rarely occurs in a previously healthy person—more commonly in the debilitated or in the diabetic subject. (2) Gangrene is very prone to follow the aspiration pneumonia, since the foreign particles rapidly undergo putrefactive changes. Of a similar nature are the cases of gangrene due to perforation of cancer of the œsophagus into the lung or into a bronchus. (3) The putrid contents of a bronchiectatic, more commonly of a tuberculous, cavity may excite gangrene in the neighboring tissues. The pressure bronchiectasis following aneurism or tumor may lead to extensive sloughing. (4) Gangrene may follow simple embolism of the pulmonary artery. More commonly, however, the embolus is derived from a part which is mortified or comes from a focus of bone disease. In typhus and in typhoid fever

gangrene of the lung may follow thrombosis of one of the larger branches of the pulmonary artery. A case occurred in my wards in October, 1897, in connection with a typhoid septicæmia. Typhoid bacilli were isolated from the lung. Lastly, gangrene of the lung may occur in conditions of debility during convalescence from protracted fever—occasionally, indeed, without our being able to assign any reasonable cause.

Morbid Anatomy.—Laennec, who first accurately described pulmonary gangrene, recognized a diffuse and a circumscribed form. The former, though rare, is sometimes seen in connection with pneumonia, more rarely after obliteration of a large branch of the pulmonary artery. It may involve the greater part of a lobe, and the lung tissue is converted into a horribly offensive greenish-black mass, torn and ragged in the centre. In the circumscribed form there is well-marked limitation between the gangrenous area and the surrounding tissue. The focus may be single or there may be two or more. The lower lobe is more commonly affected than the upper, and the peripheral more than the central portion of the lung. A gangrenous area is at first uniformly greenish brown in color; but softening rapidly takes place with the formation of a cavity with shreddy, irregular walls and a greenish, offensive fluid. The lung tissue in the immediate neighborhood shows a zone of deep congestion, often consolidation, and outside this an intense œdema. In the embolic cases the plugged artery can sometimes be found. When rapidly extending, vessels may be opened and a copious hæmorrhage ensue. Perforation of the pleura is not uncommon. The irritating decomposing material usually excites the most intense bronchitis. Embolic processes are not infrequent. There is a remarkable association in some cases between circumscribed gangrene of the lung and abscess of the brain. It has been referred to under the section on bronchiectasis.

Symptoms and Course.—Usually definite symptoms of local pulmonary disease precede the characteristic features of gangrene. These, of course, are very varied, depending on the nature of the trouble. The sputum is very characteristic. It is intensely fetid—usually profuse—and, if expectorated into a conical glass, separates into three layers—a greenish-brown, heavy sediment; an intervening thin liquid, which sometimes has a greenish or a brownish tint; and, on top, a thick, frothy layer. Spread on a glass plate, the shreddy *débris* of lung tissue can readily be picked out. Even large fragments of lung may be coughed up. Robertson, of Onancock, Va., sent me one several centimetres in length, which had been expectorated by a lad of eighteen, who had severe gangrene and recovered. Microscopically, elastic fibres are found in abundance, with granular matter, pigment grains, fatty crystals, bacteria, and leptothrix. It is stated that elastic tissue is sometimes absent, but I have never met with such an instance. The peculiar plugs of sputum which occur in bronchiectasy are not found. Blood is often present, and, as a rule, is much altered. The sputum has, in a majority of the cases, an intensely fetid odor, which is communicated to the breath and may permeate the entire room. It is much more offensive than in fetid bronchitis or in abscess of the lung. The fetor is particularly marked when there is free communication between the

gangrenous cavities and the bronchi. On several occasions I have found, post mortem, localized gangrene, which had been unsuspected during life, and in which there had been no fetor of the breath.

The physical signs, when extensive destruction has occurred, are those of cavity, but the limited circumscribed areas may be difficult to detect. Bronchitis is always present.

Among the general symptoms may be mentioned fever, usually of moderate grade; the pulse is rapid, and very often the constitutional depression is severe. But the only special features indicative of gangrene are the sputa and the fetor of the breath. The patient generally sinks from exhaustion. Fatal hæmorrhage may ensue.

Treatment.—The treatment of gangrene is very unsatisfactory. The indications, of course, are to disinfect the gangrenous area, but this is often impossible. An antiseptic spray of carbolic acid may be employed. A good plan is for the patient to use over the mouth and nose an inhaler, which may be charged with a solution of carbolic acid or with guaiacol; the latter drug has also been used hypodermically, with, it is said, happy results in removing the odor. If the signs of cavity are distinct an attempt should be made to cleanse it by direct injections of an antiseptic solution. If the patient's condition is good and the gangrenous region can be localized, surgical interference may be indicated. Successful cases have been reported. The general condition of the patient is always such as to demand the greatest care in the matter of diet and nursing.

VII. ABSCESS OF THE LUNG.

Etiology.—Suppuration occurs in the lung under the following conditions: (1) As a sequence of inflammation, either lobar or lobular. Apart from the purulent infiltration this is unquestionably rare, and even in lobar pneumonia the abscesses are of small size and usually involve, as Addison remarked, several points at the same time. On the other hand, abscess formation is extremely frequent in the deglutition and aspiration forms of lobular pneumonia. After wounds of the neck or operations upon the throat, in suppurative disease of the nose or larynx, occasionally even of the ear (Volkmann), infective particles reach the bronchial tubes by aspiration and excite an intense inflammation which often ends in abscess. Cancer of the œsophagus, perforating the root of the lung or into the bronchi, may produce extensive suppuration. The abscesses vary in size from a walnut to an orange, and have ragged and irregular walls, and purulent, sometimes necrotic, contents.

(2) Embolic, so-called metastatic, abscesses, the result of infectious emboli, are extremely common in a large proportion of all cases of pyæmia. They may occur in enormous numbers and present very definite characters. As a rule they are superficial, beneath the pleura, and often wedge-shaped. At first firm, grayish red in color, and surrounded by a zone of intense hyperæmia, suppuration soon follows with the formation of a definite abscess. The pleura is usually covered with greenish

lymph, and perforation sometimes takes place with the production of pneumothorax.

(3) Perforation of the lung from without, lodgment of foreign bodies, and, in the right lung, perforation from abscess of the liver or a suppurating echinococcus cyst are occasional causes of pulmonary abscess.

(4) Suppurative processes play an important part in chronic pulmonary tuberculosis, many of the symptoms of which are due to them.

Symptoms.—Abscess following pneumonia is easily recognized by an aggravation of the general symptoms and by the physical signs of cavity and the characters of the expectoration. Embolic abscesses cannot often be recognized, and the local symptoms are generally masked in the general pyæmic manifestations. The characters of the sputum are of great importance in determining the presence of abscess. The odor is offensive, yet it rarely has the horrible fetor of gangrene or of putrid bronchitis. In the pus fragments of lung tissue can be seen, and the elastic tissue may be very abundant. The presence of this with the physical signs rarely leaves any question as to the nature of the trouble. Embolic cases usually run a fatal course. Recovery occasionally occurs after pneumonia. In a case following typhoid fever which I saw at the Garfield Hospital, Kerr removed two ribs and found free in the pus of a localized empyema a sequestrated piece of lung, the size of the palm of the hand, which had sloughed off clearly from the lower lobe. The patient made a good recovery.

Medicinal treatment is of little avail in abscess of the lung. When well defined and superficial, an attempt should always be made to open and drain it. A number of successful cases have already been treated in this way.

VIII. NEW GROWTHS IN THE LUNGS.

Etiology and Morbid Anatomy.—While primary tumors are rare, secondary growths are not uncommon.

The primary growths of the lung are either encephaloid, scirrhus or epithelioma. Recent observations show that the last is the most common form. Sarcoma also is occasionally found as a primary growth, and still more rarely enchondroma.

The secondary growths may be of various forms. Most commonly they follow tumors in the digestive or genito-urinary organs; not infrequently also tumors of the bone. There may be encephaloid, scirrhus, epithelioma, colloid, melano-sarcoma, enchondroma, or osteoma.

Primary cancer or sarcoma usually involves only one lung. The secondary growths are distributed in both. The primary growth generally forms a large mass, which may occupy the greater part of a lung. Occasionally the secondary growths are solitary and confined chiefly to the pleura. The metastatic growths are nearly always disseminated. Occasionally they occupy a large portion of the pulmonary tissue. In a case of colloid cancer secondary to cancer of the pancreas, I found both lungs voluminous, heavy,

only slightly crepitant, and occupied by circular translucent masses, varying in size from a pea to a large walnut.

There are numerous accessory lesions in the pulmonary new growths. There may be pleurisy, either cancerous or sero-fibrinous. The effusion may be hæmorrhagic, but in 200 cases of cancer, primary or secondary, of the lungs and pleura analyzed by Moutard-Martin, hæmorrhagic effusion occurred in only 12 per cent. The tracheal and bronchial glands are usually affected, the cervical glands not infrequently, and occasionally even the inguinal.

The disease is most common in the middle period of life. The primary form affects the sexes equally, but secondary cancer is much more frequent in women than in men. The conditions which predispose to it are quite unknown. It is a remarkable fact that the workers in the Schneeberg cobalt mines are very liable to primary cancer of the lungs. It is stated that in this region a considerable proportion of all deaths in persons over forty are due to this disease.

Symptoms.—The clinical features of neoplasms of the lungs are by no means distinctive, particularly in the case of primary growths. The patient may, indeed, as noted by Walshe, present no symptoms pointing to intrathoracic disease. Among the more important symptoms are pain, particularly when the pleura is involved; dyspnœa, which is apt to be paroxysmal when due to pressure upon the trachea; cough, which may be dry and painful and accompanied by the expectoration of a dark mucoid sputum. This so-called prune-juice expectoration, which was present 10 times in 18 cases of primary cancer of the lung, was thought by Stokes to be of great diagnostic value.

In many instances there are signs of compression of the large veins, producing lividity of the face and upper extremities, or occasionally of only one arm. Compression of the trachea and bronchi may give rise to urgent dyspnœa. The heart may be pushed over to the opposite side. The pneumogastric and recurrent laryngeal nerves are occasionally involved in the growth.

Physical Signs.—The patient, according to Walshe, usually lies on the affected side. On inspection this side may be enlarged and immobile and the intercostal spaces are obliterated. This is more commonly due to the effusion than to the growth itself. The external lymph-glands may be enlarged, particularly the clavicular. The signs, on percussion and auscultation, are varied, depending much upon the presence or absence of fluid. Signs of consolidation are, of course, present; the tactile fremitus is absent and the breath-sounds are usually diminished in intensity. Occasionally there is typical bronchial breathing. Among other symptoms may be mentioned fever, which is present in a certain number of cases. Emaciation is not necessarily extreme. The duration of the disease is from six to eight months. Occasionally it runs a very acute course, as noted by Carswell. Cases are reported in which death occurred in a month or six weeks, and in one instance (Jaccoud) the patient died in a week from the onset of the symptoms.

Diagnosis.—In secondary growths this is not difficult. The development of pulmonary symptoms within a year or two after the removal of a cancer of the breast, or after the amputation of a limb for osteo-sarcoma, or the onset of similar symptoms in connection with cancer of the liver, or of the uterus, or of the rectum, would be extremely suggestive. In primary cases the unilateral involvement, the anomalous character of the physical signs, the occurrence of prune-juice expectoration, the progressive wasting, and the secondary involvement of the cervical glands are the important points in the diagnosis.

New growths are occasionally primary in the pleura (Harris, Journal of Pathology, vol. ii).

V. DISEASES OF THE PLEURA.

I. ACUTE PLEURISY.

Anatomically, the cases may be divided into dry or adhesive pleurisy and pleurisy with effusion. Another classification is into primary or secondary forms. According to the course of the disease, a division may be made into *acute* and *chronic* pleurisy, and as it is impossible, at present, to group the various forms etiologically, this is perhaps the most satisfactory division. The following forms of acute pleurisy may be considered:

I. Fibrinous or Plastic Pleurisy.

In this the pleural membrane is covered by a sheeting of lymph of variable thickness, which gives it a turbid, granular appearance, or the fibrin may exist in distinct layers. It occurs (1) as an independent affection, following cold or exposure. This form of acute plastic pleurisy without fluid exudate is not common in perfectly healthy individuals. Cases are met with, however, in which the disease sets in with the usual symptoms of pain in the side and slight fever, and there are the physical signs of pleurisy as indicated by the friction. After persisting for a few days, the friction murmur disappears and no exudation occurs. Union takes place between the membranes, and possibly the pleuritic adhesions which are found in such a large percentage of all bodies examined after death originate in these slight fibrinous pleurisies.

Fibrinous pleurisy occurs (2) as a secondary process in acute diseases of the lung, such as pneumonia, which is always accompanied by a certain amount of pleurisy, usually of this form. Cancer, abscess, and gangrene also cause plastic pleurisy when the surface of the lung becomes involved. This condition is specially associated in a large number of cases with tuberculosis. Pleural pain, stitch in the side, and a dry cough, with marked friction sounds on auscultation are the initial phenomena in many instances of phthisis. The signs are usually basic, but Burney Yeo has recently called attention to the frequency with which they occur at the apex.

II. Sero-fibrinous Pleurisy.

In a majority of cases of inflammation of the pleura there is, with the fibrin, a variable amount of fluid exudate, which produces the condition known as pleurisy with effusion.

Etiology.—For generations physicians have considered cold the potent factor in inducing pleurisy. This may be true in many cases, but modern views of serous inflammations scarcely recognize cold as anything more than a predisposing agent, which permits the action of various micro-organisms. We have not yet, however, brought all the acute pleurisies into the category of microbic affections, and the fact remains that pleurisy does follow with great rapidity a sudden wetting or a chill. Of late years an attempt has been made, particularly by French writers, to show that the majority of acute pleurisies are tuberculous. In this connection the following facts may be admitted: (1) In a large number of cases of pleurisy coming on abruptly in healthy persons the disease has been shown—(*a*) by post-mortem, in cases of accidental or sudden death, (*b*) by the subsequent history—to be tuberculous; (2) in a larger proportion of those cases which come on insidiously in persons who have been in failing health or who are delicate the disease is tuberculous from the outset; (3) the acute pleurisy, which occurs as a secondary, often a terminal, event in chronic affections, such as cirrhosis of the liver, Bright's disease, and cancer, is very frequently tuberculous. I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin. The subsequent history of cases of acute pleurisy forces us to conclude that in at least two thirds of the cases it is a curable affection. This may well be so, according to our present ideas of local tuberculous disease. Several years ago I looked over the post-mortem records of 101 successive cases which had died in my wards with pleurisy—fibrinous, sero-fibrinous, hæmorrhagic, or purulent. Of these, there were only 32 in which the pleurisy was definitely tuberculous. One of the most interesting contributions to this question has been made from the records of Henry I. Bowditch, of Boston, to whom we are indebted for so many important additions to our knowledge of pleurisy.* Of 90 cases of acute pleurisy which had been under observation between 1849 and 1879, 32 died of or had phthisis—a percentage large enough to indicate what an important *rôle* tuberculosis plays in the etiology of this disease.

Bacteriology of Acute Pleurisy.—From a bacteriological standpoint we may recognize three groups of cases of acute pleurisy: the tuberculous, the pneumococcus, and the streptococcus.

The *bacillus tuberculosis* is present in a very large proportion of all cases of primary or so-called idiopathic pleurisy. The exudate is usually sterile on cover-slips or in the culture and inoculation tests made in the ordinary way, as the bacilli are very scanty. It has been demonstrated clearly that a large amount of the exudate must be taken to make the test complete, either in cultures or in the inoculation of animals. Eichhorst

* Vincent Y. Bowditch, in Boston Medical and Surgical Journal, 1889.

found that more that 62 per cent were demonstrated as tuberculous when as much as 15 cc. of the exudate was inoculated into test animals, while less than 10 per cent of the cases showed tuberculosis when only 1 cc. of the exudate was used. This is a point to which observers should pay very special attention. Le Damany has recently in 55 primary pleurisies demonstrated the tuberculous character of all but 4. He has used large quantities of the fluid for his inoculation experiments.

The pneumococcus pleurisy is almost always secondary to a focus of inflammation in the lung. It may, however, be primary. The exudate is usually purulent and the outlook is very favorable.

The streptococcus pleurisy is the typical septic form which may occur either from direct infection of the pleura through the lung in bronchopneumonia, or in cases of streptococcus pneumonia; in other instances it follows infection of more distant parts. The acute streptococcus pleurisy is the most serious and fatal of all forms.

Among other bacilli which have been found are the staphylococcus, Friedländer's bacillus, the typhoid bacillus, and the diphtheria bacillus.

Morbid Anatomy.—In sero-fibrinous pleurisy the serous exudate is abundant and the fibrin is found on the pleural surfaces and scattered through the fluid in the form of flocculi. The proportion of these constituents varies a great deal. In some instances there is very little membranous fibrin; in others it forms thick, creamy layers and exists in the dependent part of the fluid as whitish, curd-like masses. The fluid of sero-fibrinous pleurisy is of a lemon color, either clear or slightly turbid, depending on the number of formed elements. In some instances it has a dark-brown color. The microscopical examination of the fluid shows leucocytes, occasional swollen cells, which may possibly be derived from the pleural endothelium, shreds of fibrillated fibrin, and a variable number of red blood-corpuscles. On boiling, the fluid is found to be rich in albumin. Sometimes it coagulates spontaneously. Its composition closely resembles that of blood-serum. Cholesterin, uric acid, and sugar are occasionally found. The amount of the effusion varies from ½ to 4 litres.

The lung in acute sero-fibrinous pleurisy is more or less compressed. If the exudation is limited the lower lobe alone is atelectatic; but in an extensive effusion which reaches to the clavicle the entire lung will be found lying close to the spine, dark and airless, or even bloodless—i. e., carnified.

In large exudations the adjacent organs are displaced. In large right-sided pleurisies the liver is much depressed. Rather varying statements are made with reference to the position of the heart and as to whether or not it rotates on its axis. In a number of post-mortems I have carefully studied its position, both in pneumothorax and in large effusions, and can speak with some degree of certainty on the following points: (1) Even in the most extensive left-sided exudation there is no rotation of the apex of the heart, which in no case was to the right of the mid-sternal line; (2) the relative position of the apex and base is usually maintained; in some instances the apex is lifted, in others the whole heart lies more trans-

versely; (3) the right chambers of the heart occupy the greater portion of the front, so that the displacement is rather a definite dislocation of the mediastinum, with the pericardium, to the right, than any special twisting of the heart itself; (4) the kink or twist in the inferior vena cava described by Bartels was not present in any of the cases.

Symptoms.—Prodromes are not uncommon, but the disease may set in abruptly with a chill, followed by fever and a severe pain in the side. In very many cases, however, the onset is insidious. Washbourn has called attention to the frequency with which the pneumococcus pleurisy sets in with the features of pneumonia. The pain in the side is the most distressing symptom, and is usually referred to the nipple or axillary regions. It must be remembered, however, that pleuritic pain may be felt in the abdomen or low down in the back, particularly when the diaphragmatic surface of the pleura is involved. It is lancinating, sharp, and severe, and is aggravated by cough. At this early stage, on auscultation, sometimes indeed on palpation, a dry friction rub can be detected. The fever rarely rises so rapidly as in pneumonia, and does not reach the same grade. A temperature of from 102° to 103° is an average pyrexia. It may drop to normal at the end of a week or ten days without the appearance of any definite change in the physical signs, or it may persist for several weeks. The temperature of the affected is higher than that of the sound side. Cough is an early symptom in acute pleurisy, but is rarely so distressing or so frequent as in pneumonia. There are instances in which it is absent. The expectoration is usually slight in amount, mucoid in character, and occasionally streaked with blood.

At the outset there may be dyspnœa, due partly to the fever and partly to the pain in the side. Later it results from the compression of the lung, particularly if the exudation has taken place rapidly. When, however, the fluid is effused slowly, one lung may be entirely compressed without inducing shortness of breath, except on exertion, and the patient will lie quietly in bed without evincing the slightest respiratory distress. When the effusion is large the patient usually prefers to lie upon the affected side.

Physical Signs.—*Inspection* shows some degree of immobility on the affected side, depending upon the amount of exudation, and in large effusions an increase in volume, which may appear to be much more than it really is as determined by mensuration. The intercostal spaces are obliterated. In right-sided effusions the apex beat may be lifted to the fourth interspace or be pushed beyond the left nipple, or may even be seen in the axilla. When the exudation is on the left side, the heart's impulse may not be visible; but if the effusion is large it is seen in the third and fourth spaces on the right side, and sometimes as far out as the nipple, or even beyond it.

Palpation enables us more successfully to determine the deficient movements on the affected side, and the obliteration of the intercostal spaces, and more accurately to define the position of the heart's impulse. In simple sero-fibrinous effusion there is rarely any œdema of the chest walls. It is scarcely ever possible to obtain fluctuation. Tactile fremitus is greatly diminished or abolished. If the effusion is slight there may be only en-

feeblement. The absence of the voice vibrations in effusions of any size constitutes one of the most valuable of physical signs. In children there may be much effusion with retention of fremitus. In rare cases the vibrations may be communicated to the chest walls through localized pleural adhesions.

Mensuration.—With the cyrtometer, if the effusion is excessive, a difference of from half an inch to an inch, or even, in large effusions, an inch and a half, may be found between the two sides. Allowance must be made for the fact that the right side is naturally larger than the left. With the saddle-tape the difference in expansion between the two sides can be conveniently measured.

Percussion.—Early in the disease, when the pain in the side is severe and the friction murmur evident, there may be no alteration, but with the gradual accumulation of the fluid the resonance becomes defective, and finally gives place to absolute flatness. From day to day the gradual increase in height of the fluid may be studied. In a pleuritic effusion rising to the fourth rib in front, the percussion signs are usually very suggestive. In the subclavicular region the attention is often aroused at once by a tympanitic note, the so-called Skoda's resonance, which is heard perhaps more commonly in this situation with pleural effusion than in any other condition. It shades insensibly into a flat note in the lower mammary and axillary regions. Skoda's resonance may be obtained also behind, just above the limit of effusion. The dulness has a peculiarly resistant, wooden quality, differing from that of pneumonia and readily recognized by skilled fingers. It has long been known that when the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind than it is in front, forming a parabola. The curve marking the intersection of the plane of contact of lung and fluid with the chest wall has been variously described. The "Ellis line of flatness," which Garland has verified clinically and by animal experiments, is perhaps the most characteristic. With medium-sized effusions "this line begins lowest behind, advances upward and forward in a letter-S curve to the axillary region, whence it proceeds in a straight decline to the sternum." Such a curve is present only when the patient is in the erect position, when the lung is in fairly normal condition, since then by its elastic tension it controls the position and shape of the mass of fluid, even supporting the entire weight of a considerable exudate, and when the pleuræ are free from adhesions. With larger exudates the curve flattens much, but the S can be detached with the fluid as high as the third rib. Garland emphasizes that the line can be accurately determined only by light percussion. (Garland's exhaustive work on Pneumo-dynamics.)

On the right side the dulness passes without change into that of the liver. On the left side in the nipple line it extends to and may obliterate Traube's semilunar space. If the effusion is moderate, the phenomenon of movable dulness may be obtained by marking carefully, in the sitting posture, the upper limit in the mammary region, and then in the recumbent posture, noting the change in the height of dulness. This infallible sign of fluid cannot always be obtained. In very copious exudation the

dulness may reach the clavicle and even extend beyond the sternal margin of the opposite side.

Auscultation.—Early in the disease a friction rub can usually be heard, which disappears as the fluid accumulates. It is a to-and-fro dry rub, close to the ear, and has a leathery, creaking character. There is another pleural friction sound which closely resembles, and is scarcely to be distinguished from, the fine crackling crepitus of pneumonia. This may be heard at the commencement of the disease, and also, as pointed out in 1844 by MacDonnell, Sr., of Montreal, when the effusion has receded and the pleural layers come together again.

With even a slight exudation there is weakened or distant breathing. Often inspiration and expiration are distinctly audible, though distant, and have a tubular quality. Sometimes only a puffing tubular expiration is heard, which may have a metallic or amphoric quality. Loud resonant rales accompanying this may forcibly suggest a cavity. These pseudo-cavernous signs are met with more frequently in children, and often lead to error in diagnosis. Above the line of dulness the breath-sounds are usually harsh and exaggerated, and may have a tubular quality.

The vocal resonance is usually diminished or absent. The whispered voice is said to be transmitted through a serous and not through a purulent exudate (Baccelli's sign). This author advises direct auscultation in the antero-lateral region of the chest. There may, however, be intensification—bronchophony. The voice sometimes has a curious nasal, squeaking character, which was termed by Laennec *ægophony*, from its supposed resemblance to the bleating of a goat. In typical form this is not common, but it is by no means rare to hear a curious twang-like quality in the voice, particularly at the outer angle of the scapula.

In the examination of the heart in cases of pleuritic effusion it is well to bear in mind that when the apex of the heart lies beneath the sternum there may be no impulse. The determination of the situation of the organ may rest with the position of maximum loudness of the sounds. Over the displaced organ a systolic murmur may be heard. When the lappet of lung over the pericardium is involved on either side there may be a pleuro-pericardial friction. A leucocytosis is usually present.

The *course* of acute sero-fibrinous pleurisy is very variable. After persisting for a week or ten days the fever subsides, the cough and pain disappear, and a slight effusion may be quickly absorbed. In cases in which the effusion reaches as high as the fourth rib recovery is usually slower. Many instances come under observation for the first time, after two or three weeks' indisposition, with the fluid at a level with the clavicle. The fever may last from ten to twenty days without exciting anxiety, though, as a rule, in ordinary pleurisy from cold, as we say, the temperature in cases of moderate severity is normal within eight or ten days. Left to itself the natural tendency is to resorption; but this may take place very slowly. With the absorption of the fluid there is a redux-friction crepitus, either leathery and creaking or crackling and rale-like, and for months, or even longer, the defective resonance and feeble breathing are heard at the base. Rare modes of termination are perforation and discharge through the lung,

and externally through the chest wall, examples of which have been recorded by Sahli.

A sero-fibrinous exudate may persist for months without change, particularly in tuberculous cases, and will sometimes reaccumulate after aspiration and resist all treatment. After persistence for more than twelve months, in spite of repeated tapping, a serous effusion was cured by incision without deformity of the chest (S. West). The change of the exudate into pus will be spoken of in connection with empyema. Death is a rare termination of sero-fibrinous effusion. When one pleura is full and the heart is greatly dislocated, the condition, although in a majority of cases producing remarkably little disturbance, is not without risk. *Sudden death* may occur, and its possibility under these circumstances should always be considered. I have seen two instances—one in right and the other in left sided effusion—both due, apparently, to syncope following slight exertion, such as getting out of bed. In neither case, however, was the amount of fluid excessive. Weil, who has studied carefully this accident, concludes as follows: (1) That it may be due to thrombosis or embolism of the heart or pulmonary artery, œdema of the opposite lung, or degeneration of the heart muscle; (2) such alleged causes as mechanical impediment to the circulation, owing to dislocation of the heart or twisting of the great vessels, require further investigation. Death may occur without any premonitory symptoms.

III. Purulent Pleurisy (*Empyema*).

Etiology.—Pus in the pleura is met with under the following conditions: (*a*) As a sequence of acute sero-fibrinous pleurisy. It is not always easy to say why, in certain cases, the exudate becomes purulent. It rarely does so in the acute pleurisies of healthy individuals. In children many cases are probably purulent from the onset. Aspiration, which is said to favor the occurrence of empyema, in my experience does so very rarely. (*b*) Purulent pleurisy is common as a secondary inflammation in various infectious diseases, among which scarlet fever takes the first place. It has long been known that the pleurisy supervening in the convalescence of this disease is almost always purulent. It should be remembered that it is latent in its onset, and that there may be no pulmonary symptoms. The pleurisy following typhoid fever is also usually purulent. Other infectious diseases —measles and whooping-cough—are more rarely followed by this complication. Of late years especial attention has been paid to the connection of pneumonia with empyema, and it has been shown that very many cases come on insidiously either in the course of or during convalescence from this disease; and, lastly, a limited number of tuberculous pleurisies early become purulent. (*c*) Empyema results from local causes—fracture of the rib, penetrating wounds, malignant disease of the lung or œsophagus, and, perhaps most frequently of all, the perforation of the pleura by tuberculous cavities.

The bacteriology of empyema is of great importance. A sterile exudate suggests tuberculosis. In many cases the pneumococci are present, and these cases, as a rule, run a very favorable course. The streptococci are found

most commonly in the secondary cases in connection with septic processes. In a few instances psorosperms have been present.

Morbid Anatomy.—On opening an empyema post mortem, we usually find that the effusion has separated into a clear, greenish-yellow serum above and the thick, cream-like pus below. The fluid may be scarcely more than turbid, with flocculi of fibrin through it. In the pneumococcus empyema the pus is usually thick and creamy. It usually has a heavy, sweetish odor, but in some instances—particularly those following wounds—it is fetid. In cases of gangrene of the lung or pleura the pus has a horribly stinking odor. Microscopically it has the characters of ordinary pus. The pleural membranes are greatly thickened, and present a grayish-white layer from 1 to 2 mm. in thickness. On the costal pleura there may be erosions, and in old cases fistulous communications are common. The lung may be compressed to a very small limit, and the visceral pleura also may show perforations.

Symptoms.—Purulent pleurisy may begin abruptly, with the symptoms already described. More frequently it comes on insidiously in the course of other diseases or follows an ordinary sero-fibrinous pleurisy. There may be no pain in the chest, very little cough, and no dyspnœa, unless the side is very full. Symptoms of septic infection are rarely wanting. If in a child, there is a gradually developing pallor and weakness; sweats occur, and there is irregular fever. A cough is by no means constant. The leucocytes are usually much increased; in one fatal case they numbered 115,000 per cubic millimetre.

Physical Signs.—Practically they are those already considered in pleurisy with effusion. There are, however, one or two additional points to be mentioned. In empyema, particularly in children, the disproportion between the sides may be extreme. The intercostal spaces may not only be obliterated, but may bulge. Not infrequently there is œdema of the chest walls. The network of subcutaneous veins may be very distinct. It must not be forgotten that in children the breath-sounds may be *loud and tubular* over a purulent effusion of considerable size. Whispered pectoriloquy is usually not heard in empyema (Baccelli's sign). The dislocation of the heart and the displacement of the liver are more marked in empyema than in sero-fibrinous effusion—probably, as Senator suggests, owing to the greater weight of the fluid.

A curious phenomenon associated generally with empyema, but which may occur in the sero-fibrinous exudate, is *pulsating pleurisy*, first described by MacDonnell, Sr., of Montreal. Of 42 cases 39 occurred on the left side. In all but one case the fluid was purulent. Pneumothorax may be present. There are two groups of cases, the intrapleural pulsating pleurisy and the pulsating *empyema necessitatis*, in which there is an external pulsating tumor. No satisfactory explanation has been offered how the heart impulse is thus forcibly communicated through the effusion.

Empyema is a chronic affection, which in a few instances terminates naturally in recovery, but a majority of cases, if left alone, end in death. The following are some modes of natural cure: (*a*) By absorption of the fluid. In small effusions this may take place gradually. The chest wall

sinks. The pleural layers become greatly thickened and enclose between them the inspissated pus, in which lime salts are gradually deposited. Such a condition may be seen once or twice a year in the post-mortem room of any large hospital. (*b*) By perforation of the lung. Although in this event death may take place rapidly, by suffocation, as Aretæus says, yet in cases in which it occurs gradually recovery may follow. Since 1873, when I saw a case of this kind in Traube's clinic, and heard his remarks on the subject, I have seen a number of instances of the kind and can corroborate his statement as to the favorable termination of many of them. Empyema may discharge either by opening into the bronchus and forming a fistula, or, as Traube pointed out, by producing necrosis of the pulmonary pleura, sufficient to allow the soakage of the pus through the spongy lung tissue into the bronchi. In the first way pneumothorax usually, though not always, develops. In the second way the pus is discharged without formation of pneumothorax. Even with a bronchial fistula recovery is possible. (*c*) By perforation of the chest wall—*empyema necessitatis.* This is by no means an unfavorable method, as many cases recover. The perforation may occur anywhere in the chest wall, but is, as Cruveilhier remarked, more common in front. It may be anywhere from the third to the sixth interspace, usually, according to Marshall, in the fifth. It may perforate in more than one place, and there may be a fistulous communication which opens into the pleura at some distance from the external orifice. The tumor, when near the heart, may pulsate. The discharge may persist for years. In Copeland's Dictionary is mentioned an instance of a Bavarian physician who had a pleural fistula for thirteen years and enjoyed fairly good health.

An empyema may perforate the neighboring organs, the œsophagus, peritonæum, pericardium, or the stomach. Very remarkable cases are those which pass down the spine and along the psoas into the iliac fossa, and simulate a psoas or lumbar abcess.

IV. Tuberculous Pleurisy.

This has already been considered (p. 284), and the symptoms and physical signs do not require any description other than that already given in connection with the sero-fibrinous and purulent forms.

V. Other Varieties of Pleurisy.

Hæmorrhagic Pleurisy.—A bloody effusion is met with under the following conditions: (*a*) In the pleurisy of asthenic states, such as cancer, Bright's disease, and occasionally in the malignant fevers. It is interesting to note the frequency with which hæmorrhagic pleurisy is found in cirrhosis of the liver. It occurred in the very patient in whom Laennec first accurately described this disease. While this may be a simple hæmorrhagic pleurisy, in a majority of the cases which I have seen it has been tuberculous. (*b*) Tuberculous pleurisy, in which the bloody effusion may result from the rupture of newly formed vessels in the soft exudate accom-

panying the eruption of miliary tubercles, or it may come from more slowly formed tubercles in a pleurisy secondary to extensive pulmonary disease. (*c*) Cancerous pleurisy, whether primary or secondary, is frequently hæmorrhagic. (*d*) Occasionally hæmorrhagic exudation is met with in perfectly healthy individuals, in whom there is not the slightest suspicion of tuberculosis or cancer. In one such case, a large, able-bodied man, the patient was to my knowledge healthy and strong eight years afterward. And, lastly, it must be remembered that during aspiration the lung may be wounded and blood in this way get mixed with the sero-fibrinous exudate. The condition of hæmorrhagic pleurisy is to be distinguished from hæmothorax, due to the rupture of aneurism or the pressure of a tumor on the thoracic veins.

Diaphragmatic Pleurisy.—The inflammation may be limited partly or chiefly to the diaphragmatic surface. This is often a dry pleurisy, but there may be effusion, either sero-fibrinous or purulent, which is circumscribed on the diaphragmatic surface. In these cases the pain is low in the zone of the diaphragm and may simulate that of acute abdominal disease. It may be intensified by pressure at the point of insertion of the diaphragm at the tenth rib. The diaphragm is fixed and the respiration is thoracic and short. Andral noted in certain cases severe dyspnœa and attacks simulating angina. As mentioned, the effusion is usually plastic, not serous. Serous or purulent effusions of any size limited to the diaphragmatic surface are extremely rare. Intense subjective with trifling objective features are always suggestive of diaphragmatic pleurisy.

Encysted Pleurisy.—The effusion may be circumscribed by adhesions or separated into two or more pockets or loculi, which communicate with each other. This is most common in empyema. In these cases there have usually been, at different parts of the pleura, multiple adhesions by which the fluid is limited. In other instances the recent false membranes may encapsulate the exudation on the diaphragmatic surface, for example, or the part of the pleura posterior to the mid-axillary line. The condition may be very puzzling during life, and present special difficulties in diagnosis. In some cases the tactile fremitus is retained along certain lines of adhesion. The exploratory needle should be freely used.

Interlobar Pleurisy forms an interesting and not uncommon variety. In nearly every instance of acute pleurisy the interlobular serous surfaces are also involved and closely agglutinated together, and sometimes the fluid is encysted between them. In this position tubercles are to be carefully looked for. In a case of this kind following pneumonia there was between the lower and upper and middle lobes of the right side an enormous purulent collection, which looked at first like a large abscess of the lung. These collections may perforate the bronchi, and the cases present special difficulties in diagnosis.

Diagnosis of Pleurisy.—Acute plastic pleurisy is readily recognized. In the diagnosis of pleuritic effusion the first question is, Does a fluid exudate exist? the second, What is its nature? In large effusions the increase in the size of the affected side, the immobility, the absence of tactile fremitus, together with the displacement of organs, give infallible

indications of the presence of fluid. The chief difficulty arises in effusions of moderate extent, when the dulness, the presence of bronchophony, and, perhaps, tubular breathing may simulate *pneumonia*. The chief points to be borne in mind are: (*a*) Differences in the onset and in the general characters of the two affections, more particularly the initial chill, the higher fever, more urgent dyspnœa, and the rusty expectoration, which characterize pneumonia. As already mentioned, some of the cases of pneumococcus pleurisy set in like pneumonia. (*b*) Certain physical signs—the more wooden character of the dulness, the greater resistance, and the marked diminution or the absence of tactile fremitus in pleurisy. The auscultatory signs may be deceptive. It is usually, indeed, the persistence of tubular breathing, particularly the high-pitched, even amphoric expiration, heard in some cases of pleurisy, which has raised the doubt. The intercostal spaces are more commonly obliterated in pleuritic effusion than in pneumonia. As already mentioned, the displacement of organs is a very valuable sign. Nowadays with the hypodermic needle the question is easily settled. A separate small syringe with a capacity of two drachms should be reserved for exploratory purposes, and the needle should be longer and firmer than in the ordinary hypodermic instrument. With careful preliminary disinfection the instrument can be used with impunity, and in cases of doubt the exploratory puncture should be made without hesitation. Pneumothorax is an occasional sequence. The hypodermic needle is especially useful in those cases in which there are pseudo-cavernous signs at the base. In cases, too, of massive pneumonia, in which the bronchi are plugged with fibrin, if the patient has not been seen from the outset, the diagnosis may be impossible without it.

On the left side it may be difficult to differentiate a very large pericardial from a pleural effusion. The retention of resonance at the base, the presence of Skoda's resonance toward the axilla, the absence of dislocation of the heart-beat to the right of the sternum, the feebleness of the pulse and of the heart-sounds, and the urgency of the dyspnœa, out of all proportion to the extent of the effusion, are the chief points to be considered. Unilateral hydrothorax, which is not at all uncommon in heart-disease, presents signs identical with those of sero-fibrinous effusion. Certain tumors within the chest may simulate pleural effusion. It should be remembered that many intrathoracic growths are accompanied by exudation. Malignant disease of the lung and of the pleura and hydatids of the pleura produce extensive dulness, with suppression of the breath-sounds, simulating closely effusion.

On the right side, abscess of the liver and hydatid cysts may rise high into the pleura and produce dulness and enfeebled breathing. Often in these cases there is a friction sound, which should excite suspicion, and the upper outline of the dulness is sometimes plainly convex. In a case of cancer of the kidney the growth involved the diaphragm very early, and for months there were signs of pleurisy before our attention was directed to the kidney. In all these instances the exploratory puncture should be made.

The second question, as to the nature of the fluid, is quickly decided by the use of the needle. The persistent fever, the occurrence of sweats,

a leucocytosis, and the increase in the pallor suggest the presence of pus. In children the complexion is often sallow and earthy. In protracted cases, even in children, when the general symptoms and the appearance of the patient has been most strongly suggestive of pus, the syringe has withdrawn clear fluid. On the other hand, effusions of short duration may be purulent, even when the general symptoms do not suggest it. The following statement may be made with reference to the prognostic import of the bacteriological examination of the aspirated fluid: The presence of the pneumococcus is of favorable significance, as such cases usually get well rapidly, even with a single aspiration. The streptococcus empyema is the most serious form, and even after a free drainage the patient may succumb to a general septicæmia. A sterile fluid indicates in a majority of instances a tuberculous origin.

Treatment.—At the onset the severe pain may demand leeches, which usually give relief, but a hypodermic of morphia is more effective. The Paquelin cautery may be lightly but freely applied. It is well to administer a mercurial or saline purge. Fixing the side by careful strapping with long strips of adhesive plaster, which should pass well over the middle line, drawn tightly and evenly, gives great relief, and I can corroborate the statement of F. T. Roberts as to its efficacy. Cupping, wet or dry, is now seldom employed. Blisters are of no special service in the acute stages, although they relieve the pain. The ice-bag may be used as in pneumonia. The general treatment at the early stage should be rest in bed and a liquid diet. Medicines are rarely required. A Dover's powder may be given at night. Mercurials are not indicated.

When the effusion has taken place, mustard plasters or iodine, producing slight counter-irritation, appear useful, particularly in the later stages. The following rational plan is successful in some cases. It is based upon the idea that if the blood serum is depleted or if it is kept concentrated, the liquid will be absorbed from the lymph spaces, of which the pleura is one, to equalize the loss. To do this the patient should have the daily amount of liquid food greatly restricted. If there is no fever, a meat diet, with an egg and dry bread and 8 to 10 ounces of liquid in the form of milk or water, should be given. Salt articles of food may be used, but I do not think it necessary to give, as some do, doses of salt. The second element in the treatment is the active depletion of blood serum, which is effected in the way introduced by Matthew Hay. Every morning, if the patient is robust, otherwise every second morning, from half an ounce to an ounce and a half of Epsom salts is given an hour before breakfast, in as concentrated a form as is possible. This produces copious liquid discharges. I have seen large exudations disappear rapidly when this plan was followed. By acting upon the skin and kidneys, the same end may be obtained, but with much less certainty. The vapor or hot bath may be used and an occasional dose of pilocarpin. Diuretics, such as digitalis, squills, and acetate of potash, may sometimes be required. I rarely resort, however, to diuretics or diaphoretics in the treatment of pleurisy with effusion. Iodide of potassium is of doubtful benefit. By some the salicylates are believed to be of special efficacy.

Aspiration of the fluid is the most thorough and satisfactory method and should be resorted to whenever the effusion becomes large or if it resists the ordinary methods of treatment. The credit of introducing aspiration in pleuritic effusions is due to Morrill Wyman, of Cambridge, Mass., and Henry I. Bowditch, of Boston. Years prior to Dieulafoy's work, aspiration was in constant use at the Massachusetts General Hospital and was advocated repeatedly by Bowditch. As the question is one of some historical interest, I give Bowditch's conclusions concerning aspiration, expressed nearly fifty years ago, and which practically represent the opinion of to-day: "(1) The operation is perfectly simple, but slightly painful, and can be done with ease upon any patient in however advanced a stage of the disease. (2) It should be performed forthwith in *all* cases in which there is complete filling up of one side of the chest. (3) He had determined to use it in *any* case of even *moderate* effusion lasting more than a few weeks and in which there should seem to be a disposition to resist ordinary modes of treatment. (4) He urged this practice upon the profession as a very important measure in practical medicine; believing that by this method death may frequently be prevented from ensuing either by sudden attack of dyspnœa or subsequent phthisis, and, finally, from the gradual wearing out of the powers of life or inability to absorb the fluid. (5) He believed that this operation would sometimes prevent the occurrence of those tedious cases of spontaneous evacuation of purulent fluid and those great contractions of the chest which occur after long-continued effusion and the subsequent discharge or absorption of a fluid."

There is scarcely anything to be added to-day to these observations. When the fluid reaches to the clavicle the indication for aspiration is imperative, even though the patient be comfortable and present no signs of pulmonary distress. The presence of fever is not a contra-indication; indeed, sometimes with serous exudates the temperature falls after aspiration.

The operation is extremely simple and is practically without risk. The spot selected for puncture should be either in the seventh interspace in the mid-axilla or at the outer angle of the scapula in the eighth interspace. The arm of the patient should be brought forward with the hand on the opposite shoulder, so as to widen the interspaces. The needle should be thrust in close to the upper margin of the rib, so as to avoid the intercostal artery, the wounding of which, however, is an excessively rare accident. The fluid should be withdrawn slowly. The amount will depend on the size of the exudate. If the fluid reaches to the clavicle a litre or more may be withdrawn with safety. In chronic cases of serous pleurisy after repeated tappings S. West has shown the great value of free incision and drainage. He has reported cases of recovery after effusions of fifteen and eighteen months' standing.

Symptoms and Accidents during Paracentesis.—*Pain* is usually complained of after a certain amount of fluid has been withdrawn; it is sharp and cutting in character. *Coughing* occurs toward the close, and may be severe and paroxysmal. *Pneumothorax* may follow an exploratory puncture with a hypodermic needle; it is rare during aspiration. *Subcutaneous emphysema* may develop from the point of puncture, without the production

of pneumothorax. *Albuminous expectoration* is a remarkable phenomenon described by French writers. It usually develops after the tapping, is associated with dyspnœa, and many prove suddenly fatal. *Cerebral symptoms.*—Faintness is not uncommon. Epileptic convulsions may occur either during the withdrawal or while irrigating the pleura. I have seen but a single instance. They are very difficult to explain and are regarded by most authors as of reflex origin; and lastly *sudden death* may occur either from syncope or during the convulsions.

Empyema is really a surgical affection, and I shall make only a few general remarks upon its treatment. When it has been determined by exploratory puncture that the fluid is purulent, aspiration should not be performed, except as preliminary to operation or as a temporary measure. Perhaps it is better not to have an exception to this rule, although the empyemas of children and the pneumonic empyema occasionally get well rapidly after a single tapping. It is sad to think of the number of lives which are sacrificed annually by the failure to recognize that empyema should be treated as an ordinary abscess, by free incision. The operation dates from the time of Hippocrates and is by no means serious. A majority of the cases get well, providing that free drainage is obtained, and it makes no difference practically what measures are followed so long as this indication is met. The good results in any method depend upon the thoroughness with which the cavity is drained. Irrigation of the cavity is rarely necessary unless the contents are fetid. In the subsequent treatment a point of great importance in facilitating the closure of the cavity is the distention of the lung on the affected side. This may be accomplished by the method advised by Ralston James, which has been practised with great success in the surgical wards of the Johns Hopkins Hospital. The patient daily, for a certain length of time, increasing gradually with the increase of his strength, transfers by air-pressure water from one bottle to another. The bottles should be large, holding at least a gallon each, and by the arrangement of tubes, as in the Wolff's bottle, an expiratory effort of the patient forces the water from one bottle into the other. In this way expansion of the compressed lung is systematically practised. The abscess cavity is gradually closed, partly by the falling in of the chest wall and partly by the expansion of the lung. In some instances it is necessary to resect portions of one or more ribs.

The physician is often asked, in cases of empyema with emaciation, hectic and feeble rapid pulse, whether the patient could stand the operation. Even in the most desperate cases the surgeon should never hesitate to make a free incision.

II. CHRONIC PLEURISY.

This affection occurs in two forms: (1) *Chronic pleurisy with effusion,* in which the disease may set in insidiously or may follow an acute serofibrinous pleurisy. There are cases in which the liquid persists for months or even years without undergoing any special alteration and without becom-

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7. Hercules rebel (Ephemeris XLIV) like favorable character: pure white pus "το πυον καθαρὸν καὶ λευκόν".

ing purulent. Such cases have the characters which we have described under pleurisy with effusion. (2) *Chronic dry pleurisy.* The cases are met with (*a*) as a sequence of ordinary pleural effusion. When the exudate is absorbed and the layers of the pleura come together there is left between them a variable amount of fibrinous material which gradually undergoes organization, and is converted into a layer of firm connective tissue. This process goes on at the base, and is represented clinically by a slight grade of flattening, deficient expansion, defective resonance on percussion, and enfeebled breathing. After recovery from empyema the flattening and retraction may be still more marked. In both cases it is a condition which can be greatly benefited by pulmonary gymnastics. In these firm, fibrous membranes calcification may occur, particularly after empyema. It is not very uncommon to find between the false membranes a small pocket of fluid forming a sort of pleural cyst. In the great majority of these cases the condition is one which need not cause anxiety. There may be an occasional dragging pain at the base of the lung or a stitch in the side, but patients may remain in perfectly good health for years. The most advanced grade of this secondary dry pleurisy is seen in those cases of empyema which have been left to themselves and have perforated and ultimately healed by a gradual absorption or discharge of the pus, with retraction of the side of the chest and permanent carnification of the lung. Traumatic lesions, such as gunshot wounds, may be followed by an identical condition. Post mortem, it is quite impossible to separate the layers of the pleura, which are greatly thickened, particularly at the base, and surround a compressed, airless, fibroid lung. Bronchiectasis may gradually develop, and in one remarkable case which I have seen on several occasions with Dr. Blackader, of Montreal, not only on the affected side, but also in the lower lobe of the other lung.

(*b*) *Primitive dry pleurisy.* This condition may directly follow the acute plastic pleurisy already described; but it may set in without any acute symptoms whatever, and the patient's attention may be called to it by feeling the pleural friction. A constant effect of this primitive dry pleurisy is the adhesion of the layers. This is probably an invariable result, whether the pleurisy is primary or secondary. The organization of the thin layer of exudation in a pneumonia will unite the two surfaces by delicate bands. Pleural adhesions are extremely common, and it is rare to examine a body entirely free from them. They may be limited in extent or universal. Thin fibrous adhesions do not produce any alteration in the percussion characters, and, if limited, there is no special change heard on auscultation. When, however, there is general synechia on both sides the expansile movement of the lung is considerably impaired. We should naturally think that universal adhesions would interfere materially with the function of the lungs, but practically we see many instances in which there has not been the slightest disturbance. The physical signs of total adhesion are by no means constant. It has been stated that there is a marked disproportion between the degree of expansion of the chest walls and the intensity of the vesicular murmur, but the latter is a very variable factor, and under perfectly normal conditions the breath-sounds, with very full

chest expansion, may be extremely feeble. The diaphragm phenomenon—Litten's sign—is absent.

Is there a primitive dry pleurisy which gradually leads to great thickening of the membranes, and which ultimately may invade the lung and induce cirrhotic change? Upon this question neither pathologists nor clinicians agree. I think that Sir Andrew Clark, in his Lumleian lectures at the Royal College of Physicians (1885), has made good his claim that such a disease does exist. Clinically the cases are of great interest, and should, I think, be separated, on the one hand, from the condition which follows a healed empyema or old pleurisy with effusion, and, on the other, from the rare instances of primitive cirrhosis of the lung. However, in all three states there may ultimately be an almost identical clinical picture. Anatomically in these pleuritic cases the pleura, particularly that surrounding the lower lobe, sometimes the entire membrane, is thickened, the two layers are intimately united, and fibrinous bands passing from the pleura traverse the lung tissue, sometimes dividing it in a remarkable way into sections. The bronchi may present marked dilatations, though this is not always the case, and the lung tissue is more or less sclerosed. The cases belong to the group of chronic pneumonias called by Charcot pleurogenous.

Lastly, there is a primitive dry pleurisy of tuberculous origin. In it both parietal and costal layers are greatly thickened—perhaps from 2 to 3 mm. each—and present firm fibroid, caseous masses and small tubercles, while uniting these two greatly thickened layers is a reddish-gray fibroid tissue, sometimes infiltrated with serum. This may be a local process confined to one pleura, or it may be in both. These cases are sometimes associated with a similar condition in the pericardium and peritonæum.

Occasionally remarkable vaso-motor phenomena occur in chronic pleurisy, whether simple or in connection with tuberculosis of an apex. Flushing or sweating of one cheek or dilatation of the pupil are the common manifestations. They appear to be due to involvement of the first thoracic ganglion at the top of the pleural cavity.

III. HYDROTHORAX.

Hydrothorax is a transudation of simple non-inflammatory fluid into the pleural cavities, and occurs as a secondary process in many affections. The fluid is clear, without any flocculi of fibrin, and the membranes are smooth. It is met with more particularly in connection with general dropsy, either renal, cardiac, or hæmic. It may, however, occur alone, or with only slight œdema of the feet. A child was admitted to the Montreal General Hospital with urgent dyspnœa and cyanosis, and died the night after admission. She had extensive bilateral hydrothorax, which had come on early in the nephritis of scarlet fever. In renal disease hydrothorax is almost always bilateral, but in heart affections one pleura is more commonly involved. The physical signs are those of pleural effusion, but the exudation is rarely excessive. In kidney and heart-disease, even when

there is no general dropsy, the occurrence of dyspnœa should at once direct attention to the pleura, since many patients are carried off by a rapid effusion. Post-mortem records show the frequency with which this condition is overlooked. The saline purges will in many cases rapidly reduce the effusion, but, if necessary, aspiration should repeatedly be practised.

IV. PNEUMOTHORAX (*Hydro-Pneumothorax and Pyo-Pneumothorax*).

Air alone in the pleural cavity, to which the term pneumothorax is strictly applicable, is an extremely rare condition. It is almost invariably associated with a serous fluid—hydro-pneumothorax, or with pus—pyo-pneumothorax.

Etiology.—There exists normally within the pleural cavity of an adult a negative pressure of several millimetres of mercury, due to the recoil of the distended, perfectly elastic, lung. Hence through any opening connecting the pleural cavity with the external air we should expect air to rush in until this negative pressure is relieved. To explain the absence of pneumothorax in a few cases in which it would be expected, S. West has assumed the existence of a cohesion between the pleuræ which overcomes the tendency of the chest to this condition, but this force has not as yet been satisfactorily demonstrated.

In a case of pneumothorax, if the opening causing it remain patent, the intrathoracic pressure will be that of the atmosphere, the lung will be found to have collapsed by virtue of its own elastic tension, the intercostal grooves obliterated, the heart displaced to the other side, and the diaphragm lower than normal, because the negative pressure by reason of which these organs are retained in their ordinary position has been relieved. If the opening becomes closed the intrathoracic pressure may rise above the atmospheric and the above-mentioned displacements be much increased. Some of the reasons for this rise of pressure are, the valvular action of the opening during violent expiratory efforts, the rise of temperature of the imprisoned gas, and the compression of the air by the usual effusion into the cavity.

Pneumothorax arises: (1) In perforating wounds of the chest, in which case it is sometimes associated with extensive cutaneous emphysema. It has followed exploratory puncture. Herman Biggs has reported two cases and I have seen it twice. Pneumothorax rarely follows fracture of the rib, even though the lung may be torn. (2) In perforation of the pleura through the diaphragm, usually by malignant disease of the stomach or colon. The pleura may also be perforated in cases of cancer of the œsophagus. (3) When the lung is perforated. This is by far the most common cause, and may occur: (*a*) In a normal lung from rupture of the air-vesicles during straining or even when at rest. Special attention has been called to this accident by S. West and De H. Hall. The air may be absorbed and no ill effect follows. It does not necessarily excite pleurisy, as pointed out many years ago by Gairdner, but inflammation and effusion

are the usual result. In a recent case the condition developed as the patient was going down-stairs; no effusion followed; he did not react to tuberculin. (*b*) From perforation due to local disease of the lung, either the softening of a caseous focus or the breaking of a tuberculous cavity. According to S. West, 90 per cent of all the cases are due to this cause. Less common are the cases due to septic broncho-pneumonia and to gangrene. A rare cause is the breaking of a hæmorrhagic infarct in chronic heart-disease, of which I met an instance a few years ago. (*c*) Perforation of the lung from the pleura, which arises in certain cases of empyema and produces a pleuro-bronchial fistula. (*d*) Spontaneously, by the development in pleural exudates of the gas bacillus (*B. aërogenes capsulatus* Welch).

Pneumothorax occurs chiefly in adults, though cases are met with in very young children. It is more frequent in males than in females.

Morbid Anatomy.—If a trocar or blow-pipe is inserted between the ribs, there may be a jet of air of sufficient strength to blow out a lighted match. On opening the thorax the mediastinum and pericardium are seen to be pushed, or rather, as Douglas Powell pointed out, drawn over to the opposite side; but, as before mentioned, the heart is not rotated, and the relation of its parts is maintained much as in the normal condition. A serous or purulent fluid is usually present, and the membranes are inflamed. The cause of the pneumothorax can usually be found without difficulty. In the great majority of instances it is the perforation of a tuberculous cavity or a breaking of a superficial caseous focus. The orifice of rupture may be extremely small. In chronic cases there may be a fistula of considerable size communicating with the bronchi. The lung is usually compressed and carnified.

Symptoms.—The onset is usually sudden and characterized by severe pain in the side, urgent dyspnœa, and signs of general distress, as indicated by slight lividity and a very rapid and feeble pulse. There may, however, be no urgent symptoms, particularly in cases of long-standing phthisis. On more than one occasion I have found, post mortem, a pneumothorax which was unsuspected during life. West states that even in healthy adults this latent pneumothorax may occasionally occur.

A remarkable recurrent variety has been described by S. West, Goodhart, and Furney. In Goodhart's case the pneumothorax developed first in one side and then in the other.

The *physical signs* are very distinctive. *Inspection* shows marked enlargement of the affected side with immobility. The heart impulse is usually much displaced. On *palpation* the fremitus is greatly diminished or more commonly abolished. On *percussion* the resonance may be tympanitic or even have an amphoric quality. This, however, is not always the case. It may be a flat tympany, resembling Skoda's resonance. In some instances it may be a full, hyperresonant note, like emphysema; while in others—and this is very deceptive—there is dulness. These extreme variations depend doubtless upon the degree of intrapleural tension. On several occasions I have known an error in diagnosis to result from ignorance of the fact that, in certain instances, the percussion note

may be "muffled, toneless, almost dull" (Walshe). There is usually dulness at the base from effused fluid, which can readily be made to change the level by altering the position of the patient. Movable dulness can be obtained much more readily in pneumothorax than in a simple pleurisy. On *auscultation* the breath-sounds are suppressed. Sometimes there is only a distant feeble inspiratory murmur of marked amphoric quality. The contrast between the loud exaggerated breath-sounds on the normal side and the absence of the breath-sounds on the other is very suggestive. The rales have a peculiar metallic quality, and on coughing or deep inspiration there may be what Laennec termed the metallic tinkling. The voice, too, has a curious metallic echo. What is sometimes called the coin-sound, termed by Trousseau the *bruit d'airain*, is very characteristic. To obtain it the auscultator should place one ear on the back of the chest wall while the assistant taps one coin on another on the front of the chest. The metallic echoing sound which is produced in this way is one of the most constant and characteristic signs of pneumothorax. And, lastly, the Hippocratic succussion may be obtained when the auscultator's head is placed upon the chest while the patient's body is shaken. A splashing sound is produced, which may be audible at a distance. A patient may himself notice it in making abrupt changes in posture. Of other symptoms displacement of organs is most constant. As already mentioned, the heart may be drawn over to the opposite side, and the liver greatly displaced, so that its upper surface is below the level of the costal margin, a degree of dislocation never seen in simple effusion.

The *diagnosis* of pneumothorax rarely offers any difficulty, as the signs are very characteristic. In cases in which the percussion note is dull the condition may be mistaken for effusion. I made this mistake in a case of pulsating pleurisy, in which the pneumothorax followed heavy lifting, and it was not until several days later, after some of the fluid had been withdrawn, that a tympanitic note developed. Diaphragmatic hernia following a crush or other accident may closely simulate pneumothorax.

In cases of very large phthisical cavities with tympanitic percussion resonance and rales of an amphoric, metallic quality, the question of pneumothorax is sometimes raised. In those rare instances of total excavation of one lung the amphoric and metallic phenomena may be most intense, but the absence of dislocation of the organs, of the succussion splash, and of the coin-sound suffice to differentiate this condition. While this is true in the great majority of cases, I have recently heard the *bruit d'airain* over large cavities of the right upper lobe. The condition of pyo-pneumothorax subphrenicus may simulate closely true pneumothorax.

The *prognosis* in cases of pneumothorax depends largely upon the cause. S. West gives a mortality of 70 per cent. The tuberculous cases usually die within a few weeks. Of 39 cases, 29 died within a fortnight (West); 10 patients died on the first day, 2 within twenty and thirty minutes respectively of the attack. Pneumothorax developing in a healthy individual often ends in recovery. There are tuberculous cases in which the pneumothorax, if occurring early, seems to arrest the progress of the tuberculosis. This appeared to be the case in a man with chronic pneumothorax

who was under my care in Philadelphia for between three and four years. It may be a chronic condition, as in the case just mentioned, and a fair measure of health may be enjoyed.

Treatment.—Practically these cases should be dealt with as ordinary pleurisy with effusion. Of course, when pneumothorax develops in advanced phthisis the indication is to relieve the pain and distress either by morphia or chloroform; but in cases which develop early the fluid should be withdrawn by aspiration, or, if purulent, permanent drainage should be obtained. Even when the condition has seemed to be most desperate I have known recovery to take place after thorough drainage of the sac. Portions of ribs may have to be excised, and during convalescence it is well for the patient to practise expansion of the lung in the manner already mentioned. There are cases of pneumothorax in phthisis in which the general condition is so good and the inconvenience so slight that to let well enough alone seems the best course. In such an occasional aspiration may be performed if the fluid increases. In some of the instances the mere tapping of the chest with a fine needle, so as to allow the escape of some of the air, seems to give relief by reducing the intrathoracic pressure. Good results are stated to have followed the method introduced by Potain, of replacing the air and fluid within the thorax by sterilized air.

V. AFFECTIONS OF THE MEDIASTINUM.

(1) **Simple Lymphadenitis.**—In all inflammatory affections of the bronchi and of the lungs the groups of lymph-glands in the mediastinum become swollen. In the bronchitis of measles, for example, and in simple broncho-pneumonia the bronchial glands are large and infiltrated, the tissue is engorged and œdematous, sometimes intensely hyperæmic. Much stress has been laid by some writers on this enlargement of the glands in the posterior mediastinum, and De Mussy held that it was an important factor in inducing paroxysms of whooping-cough. They may attain a size sufficient to induce dulness beneath the manubrium and in the upper part of the interscapular regions behind, though this is often difficult to determine. In reality the glands lie chiefly upon the spine, and unless those which are deep in the root of the lung are large enough to induce compression of the adjacent lung tissue, I doubt if the ordinary bronchial adenopathy ever can be determined by percussion in the upper interscapular region. I have never met with an instance in which the compression of either bronchus seemed to have resulted from the glands, however large. Tuberculous affection of these glands has already been considered.

(2) **Suppurative Lymphadenitis.**—Occasionally abscess in the bronchial or tracheal lymph-glands is found. It may follow the simple adenitis, but is most frequently associated with the presence of tubercle. The liquid portion may gradually become absorbed and the inspissated contents undergo calcification. Serious accidents occasionally occur, as perforation into the œsophagus or into a bronchus, or in rare instances, as in the case

reported by Sidney Phillips, perforation of the aorta, as well as a bronchus, which, it is remarkable to say, did not prove fatal rapidly, but caused repeated attacks of hæmoptysis during a period of sixteen months.

(3) **Tumors; Cancer and Sarcoma.**—In Hare's elaborate study of 520 cases of disease of the mediastinum * there were 134 cases of cancer, 98 cases of sarcoma, 21 cases of lymphoma, 7 cases of fibroma, 11 cases of dermoid cysts, 8 cases of hydatid cysts, and instances of lipoma, gumma, and enchondroma. From this we see that cancer is the most common form of growth. The tumor occurred in the anterior mediastinum alone in 48 of the cases of cancer and in 33 of the cases of sarcoma. There are three chief points of origin, the thymus, the lymph-glands, and the pleura and lung. Sarcoma is more frequently primary than cancer. Males are more frequently affected than females. The age of onset is most commonly between thirty and forty.

Symptoms.—The signs of mediastinal tumor are those of intrathoracic pressure. *Dyspnœa* is one of the earliest and most constant symptoms, and may be due either to pressure on the trachea or on the recurrent laryngeal nerves. It may indeed be cardiac, due to pressure upon the heart or its vessels. In a few cases it results from the pleural effusion which so frequently accompanies intrathoracic growths. Associated with the dyspnœa is a cough, often severe and paroxysmal in character, with the brazen quality of the so-called aneurismal cough when a recurrent nerve is involved. The voice may also be affected from a similar cause. Pressure on the vessels is common. The superior vena cava may be compressed and obliterated, and when the process goes on slowly the collateral circulation may be completely effected. Less commonly the inferior vena cava or one or other of the subclavian veins is compressed. The arteries are much less rarely obstructed. It is remarkable how little the aorta may be involved, though entirely surrounded by a sarcomatous or cancerous mass. There may be dysphagia, due to compression of the œsophagus. In rare instances there are pupillary changes, either dilatation or contraction, due to involvement of the sympathetic.

Physical Signs.—On inspection there may be orthopnœa and marked cyanosis of the upper part of the body. In such instances, if of long duration, there are signs of collateral circulation and the superficial mammary and epigastric veins are enlarged. In these cases of chronic obstruction the finger-tips may be clubbed. There may be bulging of the sternum or the tumor may erode the bone and form a prominent subcutaneous growth. The rapidly growing lymphoid tumors more commonly than others perforate the chest wall. In 4 of 13 cases of Hodgkin's disease, there was mediastinal growth, and in 3 instances the sternum was eroded and perforated. The perforation may be on one side of the breast-bone. The projecting tumor may pulsate; the heart may be dislocated and its impulse much out of place. Contraction of one side of the thorax has been noted in a few instances. On palpation the fremitus is absent wherever the tumor reaches the chest wall. If pulsating, it rarely has the forcible,

* Fothergillian Prize Essay of the Medical Society of London, Philadelphia, 1889.

heaving impulse of an aneurismal sac. On auscultation there is usually silence over the dull region. The heart-sounds are not transmitted and the respiratory murmur is feeble or inaudible, rarely bronchial. Vocal resonance is, as a rule, absent. Signs of pleural effusion occur in a great many instances of mediastinal growth, and in doubtful cases the aspirator needle should be used.

Tumors of the anterior mediastinum originate usually in the thymus; the sternum is pushed forward and often eroded. The growth may be felt in the suprasternal fossa; the cervical glands are usually involved. The pressure symptoms are chiefly upon the venous trunks. Dyspnœa is a prominent feature.

Intrathoracic tumors in the middle and posterior mediastinum originate most commonly in the lymph-glands. The symptoms are out of all proportion to the physical signs; there is urgent dyspnœa and cough, which is sometimes loud and ringing. The pressure symptoms are chiefly upon the gullet, the recurrent laryngeal, and sometimes upon the azygos vein.

In a third group, tumors originating in the pleura and the lung, the pressure symptoms are not so marked. Pleural exudate is very much more common; the patient becomes anæmic and emaciation is rapid. There may be secondary involvement of the lymph-glands in the neck. For a discussion of the symptomatology of these different groups, see Pepper and Stengel, Transactions of the Association of American Physicians, vol. x.

The *diagnosis* of mediastinal tumor from aneurism is sometimes extremely difficult. An interesting case reported and figured by Sokolosski, in Bd. 19 of the Deutsches Archiv für klinische Medicin, in which Oppolzer diagnosed aneurism and Skoda mediastinal tumor, illustrates how in some instances the most skilful of observers may be unable to agree. Scarcely a sign is found in aneurism which may not be duplicated in mediastinal tumor. This is not strange, since the symptoms in both are largely due to pressure. The time element is important. If a case has persisted for more than eighteen months the disease is probably aneurism. There are, however, exceptions to this. By far the most valuable sign of aneurism is the diastolic shock so often to be felt, and in a majority of cases to be heard, over the sac. This is rarely, if ever, present in mediastinal growths, even when they perforate the sternum and have communicated pulsation. Tracheal tugging is rarely present in tumor. Another point of importance is that a tumor, advancing from the mediastinum, eroding the sternum and appearing externally, if aneurismal, has forcible, heaving, and distinctly expansile pulsations. The radiating pain in the back and arms and neck is rather in favor of aneurism, as is also a beneficial influence on it of iodide of potassium.

The frequency of pleural effusion in connection with mediastinal tumor is to be constantly borne in mind. It may give curiously complex characters to the physical signs—characters which are profoundly modified after aspiration of the liquid.

(4) **Abscess of the Mediastinum.**—Hare collected 115 cases of mediastinal abscess, in 77 of which there were details sufficient to permit the

analysis. Of these cases the great majority occurred in males. Forty-four were instances of acute abscess. The anterior mediastinum is most commonly the seat of the suppuration. The cases are most frequently associated with trauma. Some have followed erysipelas or occurred in association with eruptive fevers. Many cases, particularly the chronic abscesses, are of tuberculous origin. Of *symptoms*, pain behind the sternum is the most common. It may be of a throbbing character, and in the acute cases is associated with fever, sometimes with chills and sweats. If the abscess is large there may be dyspnœa. The pus may burrow into the abdomen, perforate through an intercostal space, or it may erode the sternum. Instances are on record in which the abscess has discharged into the trachea or œsophagus. In many cases, particularly of chronic abscess, the pus becomes inspissated and produces no ill effect. The *physical signs* may be very indefinite. A pulsating and fluctuating tumor may appear at the border of the sternum or at the sternal notch. The absence of *bruit*, of the diastolic shock, and of the expansile pulsation usually enables a correct diagnosis to be made. When in doubt a fine hypodermic needle may be inserted.

(5) **Indurative Mediastino-Pericarditis.**—Harris has recently reviewed the subject. In one form there is adherent pericardium and great increase in the fibrous tissues of the mediastinum; in another there is adherent pericardium with union to surrounding parts, but very little mediastinitis; in a third the pericardium may be uninvolved. The disease is rare; of 22 cases 17 were in males; only 2 were above thirty years of age. The symptoms are essentially those of that form of adhesive pericardium which is associated with great hypertrophy and dilatation of the heart, and in which the patients present a picture of cyanosis, dyspnœa, anasarca, etc. The pulsus paradoxus, described by Kussmaul, is not distinctive. Occasionally there is also a proliferative peritonitis. Mediastinal friction is sometimes heard in patients with adhesive mediastino-pericarditis—dry, coarse, crackling rales heard along the sternum, particularly when the arms are raised.

(6) **Miscellaneous Affections.**—In Hare's monograph there were 7 instances of fibroma, 11 cases of dermoid cyst, 8 cases of hydatid cyst, and cases of lipoma and gumma.

(7) **Emphysema of the Mediastinum.**—Air in the cellular tissues of the mediastinum is met with in cases of trauma, and occasionally in fatal cases of diphtheria and in whooping-cough. It may extend to the subcutaneous tissues. Champneys has called attention to its frequency after tracheotomy, in which, he says, the conditions favoring the production are division of the deep fascia, obstruction in the air-passages, and inspiratory efforts. The deep fascia, he says, should not be raised from the trachea. It is often associated with pneumothorax. The condition seems by no means uncommon. Angel Money found it in 16 of 28 cases of tracheotomy, and in 2 of these pneumothorax also was present.

SECTION VII.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASES OF THE PERICARDIUM.

1. PERICARDITIS.

PERICARDITIS is the result of infective processes, primary or secondary, or arises by extension of inflammation from contiguous organs.

Etiology.—*Primary*, so-called idiopathic, inflammation of this membrane is rare; but cases are met with, most commonly in children, in which there is no evidence of rheumatism or of other conditions with which the disease is usually associated.

Pericarditis from injury usually comes under the care of the surgeon in connection with the primary wound. Interesting cases are those in which the traumatism is from within, due to the passage of some foreign body—such as a needle, a pin, or a bone—through the œsophagus into the pericardium.

As a *secondary* process pericarditis is met with in the following affections:—(*a*) A majority of the cases occur in connection with rheumatism. The percentage given by different authors ranges from thirty to seventy. The articular trouble may be slight or, indeed, the disease may be associated with acute tonsillitis in rheumatic subjects. Cases are recorded in which the pericarditis has preceded the articular disease. (*b*) Septic processes rank next to rheumatism. In the acute necrosis of bone and puerperal fever it is not uncommon. (*c*) Tuberculosis, in which the disease may be primary or part of a general involvement of the serous sacs or associated with extensive pulmonary disease. (*d*) Eruptive fevers.—In children, the disease is not infrequent after scarlatina. It is rarely met with in measles, small-pox, or typhoid fever. In other infective diseases, such as diphtheria and pneumonia, it is rare. Pericarditis sometimes complicates chorea; it was present in 19 of 73 autopsies which I collected; in only 8 of these was arthritis present. (*e*) Certain altered conditions of the system seem to render the pericardium more susceptible to infection. Of these gout takes the first place. In chronic Bright's disease pericarditis is by no means rare. The *pericardite brightique* of the French forms one of the most important groups of the disease in persons over fifty years of age, most frequently

accompanying the chronic interstitial form of nephritis. Pericarditis has been met with also in scurvy and diabetes.

Pericarditis by extension of disease from contiguous organs. In pleuro-pneumonia it forms one of the most serious complications, and was present in 5 cases of 100 post mortems in this disease which I made at the Montreal General Hospital. It is most often met with in the pleuro-pneumonia of children and of alcoholics. The association with simple pleurisy is much less common. In ulcerative endocarditis, purulent myocarditis, and in aneurism of the aorta pericarditis is occasionally found. It may also result from extension of disease from the bronchial glands, the ribs, sternum, vertebræ, and even from the abdominal viscera. Of 100 consecutive cases at the Boston City Hospital analyzed by Sears, in 54 the exudate was dry, in 41 serous, in 4 hæmorrhagic, and in 5 purulent. Thirty-four cases showed signs of old valvular disease; rheumatism was a factor in 51; pneumonia in 18; and in 7 chronic nephritis. Of the 100 cases 43 died.

Pericarditis occurs at all ages. Cases are reported in the fœtus. In the new-born it may result from septic infection through the navel. Throughout childhood the incidence of rheumatism and scarlet fever makes it a frequent affection, whereas late in life it is most often associated with tuberculosis, Bright's disease, and gout. Males are somewhat more frequently attacked than females. Climatic and seasonal influences have been mentioned by some writers. The so-called epidemics of pericarditis have been outbreaks of pneumonia with this as a frequent complication.

Anatomically as well as clinically the disease may be considered under the following divisions:

1. Acute, plastic, or dry pericarditis.
2. Pericarditis with effusion—sero-fibrinous, hæmorrhagic, or purulent.
3. Chronic adhesive pericarditis (adherent pericardium).

Acute Plastic Pericarditis.—This, the most common form, occurs usually as a secondary process, and is distinguished by the small amount of fluid exudation, which does not, as in the next variety, give special characters to the disease. It is a benign form and never of itself proves fatal.

Anatomically it may be partial or general. In the mildest grades the serous membrane looks lustreless and roughened. This is due to the presence of a thin fibrinous sheeting, which can be lifted with the knife, showing the membrane beneath to be injected or in places ecchymotic. As the fibrinous sheeting increases in thickness the constant movement of the adjacent surfaces gives to it sometimes a ridge-like, at others a honeycombed appearance. With more abundant fibrinous exudation the membranes present an appearance resembling buttered surfaces which have been drawn apart. The fibrin is in long shreds, and the heart presents a curiously shaggy appearance—the so-called hairy heart of old writers—*cor villosum.*

In mild grades the subjacent muscle looks normal; but in the more prolonged and severe cases there is myocarditis, and for 2 or 3 mm. beneath the visceral layer the muscle presents a pale, turbid appearance. Many of these acute cases are tuberculous; covered by the layers of lymph the granulations are easily overlooked in a superficial examination.

Slight fluid exudation is invariably present, entangled in the meshes of fibrin, but there may be very thick fibrinous layers without much serous effusion.

Symptoms.—The majority of cases of simple plastic pericarditis, like those of simple endocarditis, present no symptoms, and unless sought for there are no objective signs indicating its existence. In the post-mortem room it is not uncommon to find it in cases in which its presence has been unsuspected during life.

Pain is a variable symptom, not usually intense, and in this form rarely excited by pressure. It is more marked in the early stage, and may be referred either to the præcordia or to the region of the xiphoid cartilage. Instances are recorded of pain of an aggravated and most distressing character resembling angina. Fever is usually present, but it is not always easy to say how much depends upon the primary febrile affection, and how much upon the pericarditis. It is as a rule not high, rarely exceeding 102.5°. In rheumatic cases hyperpyrexia has been observed.

Physical Signs.—*Inspection* is negative; *palpation* may reveal the presence of a distinct fremitus caused by the rubbing of the roughened pericardial surfaces. This is usually best marked over the right ventricle. It is not always to be felt, even when the friction sound on auscultation is loud and clear. *Auscultation:* The friction sound, due to the movement of the pericardial surfaces upon each other, is one of the most distinctive of physical signs. It is double, corresponding to the systole and diastole; but the synchronism with the heart-sounds is not accurate, and the to-and-fro murmur usually outlasts the time occupied by the first and second sound. In rare instances the friction is single; more frequently it appears to be triple in character—a sort of canter rhythm. The sounds have a peculiar rubbing, grating quality, characteristic when once recognized, and rarely simulated by endocardial murmurs. Sometimes instead of grating there is a creaking quality—the *bruit de cuir neuf*—the new-leather murmur of the French. The pericardial friction appears superficial, very close to the ear, and is usually intensified by pressure with the stethoscope. It is best heard over the right ventricle, the part of the heart which is most closely in contact with the front of the chest—that is, in the fourth and fifth interspaces and adjacent portions of the sternum. There are instances in which the friction is most marked at the base, over the aorta, and at the superior reflection of the pericardium. Occasionally it is best heard at the apex. It may be limited and heard over a very narrow area, or it may be transmitted up and down the sternum. There are, however, no definite lines of transmission as in the endocardial murmur. An important point is the variability of the sounds, both in position and quality; they may be heard at one visit and not at another. The maximum of intensity will be found to vary with position.

Diagnosis.—There is rarely any difficulty in determining the presence of a dry pericarditis, for the friction sounds are distinctive. The double murmur of aortic incompetency may simulate closely the to-and-fro pericardial rub. I recall one instance at least in which this mistake was made. The constant character of the aortic murmur, the direction of trans-

mission, the phenomena in the arteries, and the associated conditions of the disease should be sufficient to prevent this error.

I have never known an instance in which pericarditis was mistaken for acute endocarditis, though writers refer to such, and give the differential diagnosis in the two affections. The only possible mistake could be made in those rare instances of single soft, systolic, pericardial friction.

Pleuro-pericardial friction is very common, and may be associated with endo-pericarditis, particularly in cases of pleuro-pneumonia. It is frequent, too, in phthisis. It is best heard over the left border of the heart, and is much affected by the respiratory movement. Holding the breath or taking a deep inspiration may annihilate it. The rhythm is not the simple to-and-fro diastolic and systolic, but the respiratory rhythm is superadded, usually intensifying the murmur during expiration and lessening it on inspiration. In phthisis there are instances in which, with the friction, a loud systolic click is heard, due to the compression of a thin layer of lung and the expulsion of a bubble of air from a small softening focus or from a bronchus.

And, lastly, it is not very uncommon, in the region of the apex beat, to hear a series of fine crepitant sounds, systolic in time, often very distinct, suggestive of pericardial adhesions, but heard too frequently for this cause.

Course and Termination.—Simple fibrinous pericarditis never kills, but it occurs so often in connection with serious affections that we have frequent opportunities to see all stages of its progress. In the majority of cases the inflammation subsides and the thin fibrinous laminæ gradually become converted into connective tissue, which unites the pericardial leaves firmly together. In other instances the inflammation progresses, with increase of the exudation, and the condition is changed from a "dry" to a "moist" pericarditis, or the pericarditis with effusion.

In a few instances—probably always tuberculous—the simple plastic pericarditis becomes chronic, and great thickening of both visceral and parietal layers is gradually induced.

Pericarditis with Effusion.—Though commonly a direct sequence of the dry or plastic pericarditis, of which it is sometimes called the second stage, this form presents special features and deserves separate consideration. It is found most frequently in association with acute rheumatism, tuberculosis, and septicæmia, and sets in usually with the symptoms above described, namely, præcordial pain, with slight fever or a distinct chill.

In children the disease may, like pleurisy, come on without local symptoms, and, after a week or two of failing health, slight fever, shortness of breath, and increasing pallor, the physician may find, to his astonishment, signs of most extensive pericardial effusion. These latent causes are often tuberculous. W. Ewart has called special attention to latent and ephemeral pericardial effusions, which he thinks are often of short duration and of moderate size, with an absence of the painful features of pericarditis. The effusion may be sero-fibrinous, hæmorrhagic, or purulent. The amount varies from 200 or 300 cc. to 2 litres. In the cases of sero-fibrinous exudation the pericardial membranes are covered with thick, creamy fibrin, which

may be in ridges or honeycombed, or may present long, villous extensions. The parietal layer may be several millimetres in thickness and may form a firm, leathery membrane. The hæmorrhagic exudation is usually associated with tuberculous, or with cancerous pericarditis, or with the disease in the aged. The lymph is less abundant, but both surfaces are injected and often show numerous hæmorrhages. Thick, curdy masses of lymph are usually found in the dependent part of the sac. In the purulent effusion the fluid has a creamy consistency, particularly in tuberculosis. In many cases the effusion is really sero-purulent, a thin, turbid exudation containing flocculi of fibrin.

The pericardial layers are greatly thickened and covered with fibrin. When the fluid is pus, they present a grayish, rough, granular surface. Sometimes there are distinct erosions on the visceral membrane. The heart muscle in these cases becomes involved to a greater or less extent, and on section, the tissue, for a depth of from 2 to 3 mm., is pale and turbid, and shows evidence of fatty and granular change. Endocarditis coexists frequently, but rarely results from the extension of the inflammation through the wall of the heart.

Symptoms.—Even with copious effusion the onset and course may be so insidious that no suspicion of the true nature of the disease is aroused.

As in the simple pericarditis, pain may be present, either sharp and stabbing or as a sense of distress and discomfort in the cardiac region. It is more frequent with effusion than in the plastic form. Pressure at the lower end of the sternum usually aggravates it. Dyspnœa is a common and important symptom, one which, perhaps, more than any other, excites suspicion of grave disorder and leads to careful examination of heart and lungs. The patient is restless, lies upon the left side or, as the effusion increases, sits up in bed. Associated with the dyspnœa is in many cases a peculiarly dusky, anxious countenance. The pulse is rapid, small, sometimes regular, and may present the characters known as *pulsus paradoxus*, in which during each inspiration the pulse-beat becomes very weak or is lost. These symptoms are due, in great part, to the direct mechanical effect of the fluid within the pericardium which embarrasses the heart's action. Other pressure effects are distention of the veins of the neck, dysphagia, which may be a marked symptom, and irritative cough from compression of the trachea. Aphonia is not uncommon, owing to compression or irritation of the recurrent laryngeal as it winds round the aorta. Another important pressure effect is exercised upon the left lung. In massive effusion the pericardial sac occupies such a large portion of the antero-lateral region of the left side that the condition has frequently been mistaken for pleurisy. Even in moderate grades the left lung is somewhat compressed. This is an additional element in the production of the dyspnœa.

Great restlessness, insomnia, and in the later stages low delirium and coma are symptoms in the more severe cases. Delirium and marked cerebral symptoms are associated with the hyperpyrexia of rheumatic cases, but apart from the ordinary delirium there may be peculiar mental symptoms. The patient may become melancholic and show suicidal tendencies.

In other cases the condition resembles closely delirium tremens. Sibson, who has specially described this condition, states that the majority of such cases recover. Chorea may also occur, as was pointed out by Bright. Epilepsy is a rare complication which has occurred during paracentesis.

Physical Signs.—*Inspection.*—In children the præcordia bulges and with copious exudation the antero-lateral region of the left chest becomes enlarged. The intercostal spaces are prominent and there may be marked œdema of the wall. The epigastrium may be more prominent. Perforation externally through a space is very rare. Owing to the compression of the lung, the expansion of the left side is greatly diminished. The diaphragm and left lobe of the liver may be pushed down and may produce a distinct prominence in the epigastric region.

Palpation.—A gradual diminution and final obliteration of the cardiac shock is a striking feature in progressive effusion. The position of the apex beat is not constant. In large effusions it is usually not felt. In children as the fluid collects the pulsation may be best seen in the fourth space, but this may not be the apex itself. Ewart maintains that the position of the apex beat is unaltered, or even depressed. The pericardial friction may lessen with the effusion, though it often persists at the base when no longer palpable over the right ventricle, or may be felt in the erect and not in the recumbent posture. Fluctuation can rarely, if ever, be detected.

Percussion gives most important indications. The gradual distention of the pericardial sac pushes aside the margins of the lungs so that a large area comes in contact with the chest wall and gives a greatly increased percussion dulness. The form of this dulness is irregularly pear-shaped; the base or broad surface directed downward and the stem or apex directed upward toward the manubrium. A valuable sign, to which Rotch called attention, is the absence of resonance in the right fifth intercostal space. In the left infrascapular area there may be a patch of diminished resonance or even flatness (Ewart).

Auscultation.—The friction sound heard in the early stages may disappear when the effusion is copious, but often persists at the base or at the limited area of the apex. It may be audible in the erect and not in the recumbent posture. With the absorption of the fluid the friction returns. One of the most important signs is the gradual weakening of the heart-sounds, which with the increase in the effusion may become so muffled and indistinct as to be scarcely audible. The heart's action is usually increased and the rhythm disturbed. Occasionally a systolic endocardial murmur is heard. Early and persistent accentuation of the pulmonary second sound may be present (Warthin).

Important accessory signs in large effusion are due to pressure on the left lung. The antero-lateral margin of the lower lobe is pushed aside and in some instances compressed, so that percussion in the axillary region, in and just below the transverse nipple line, gives a modified percussion note, usually a flat tympany. Variations in the position of the patient may change materially this modified percussion area, over which on auscultation there is either feeble or tubular breathing.

Course.—Cases vary extremely in the rapidity with which the effusion

takes place. In every instance, when a pericardial friction murmur has been detected, the practitioner should immediately outline with care—using the aniline pencil or nitrate of silver—the upper and lateral limits of cardiac dulness, since he will in this way have certain positive guides in determining the rate and grade of the effusion. In many instances the exudation is slight in amount, reaches a maximum within forty-eight hours, and then gradually subsides. In other instances the accumulation is more gradual and progressive, increasing for several weeks. To such cases the term chronic has been applied. The rapidity with which a sero-fibrinous effusion may be absorbed is surprising. The possibility of the absorption of a purulent exudate is shown by the cases in which the pericardium contains semi-solid grayish masses in all stages of calcification. With sero-fibrinous effusion, if moderate in amount, recovery is the rule, with inevitable union, however, of the pericardial layers. In some of the septic cases there is a rapid formation of pus and a fatal result may follow in three or four days. More commonly, when death occurs with large effusion, it is not until the second or third week and takes place by gradual asthenia.

Prognosis.—In the sero-fibrinous effusions the outlook is good, and a large majority of all the rheumatic cases recover. The purulent effusions are, of course, more dangerous; the septic cases are usually fatal, and recovery is rare in the slow, insidious tuberculous forms.

Diagnosis.—Probably no serious disease is so frequently overlooked by the practitioner. Post-mortem experience shows how often pericarditis is not recognized, or goes on to resolution and adhesion without attracting notice. In a case of rheumatism, watched from the outset, with the attention directed daily to the heart, it is one of the simplest of diseases to diagnose; but when one is called to a case for the first time and finds perhaps an increased area of præcordial dulness, it is often very hard to determine with certainty whether or not effusion is present.

The difficulty usually lies in distinguishing between dilatation of the heart and pericardial effusion. Although the differential signs are simple enough on paper, it is notoriously difficult in certain cases, particularly in stout persons, to say which of the conditions exists. The points which deserve attention are:

(*a*) The character of the impulse, which in dilatation, particularly in thin-chested people, is commonly visible and wavy.

(*b*) The shock of the cardiac sounds is more distinctly palpable in dilatation.

(*c*) The area of dulness in dilatation rarely has a triangular form; nor does it, except in cases of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces *without visible or palpable impulse.* An upper limit of dulness shifting with change of position speaks strongly for effusion.

(*d*) In dilatation the heart-sounds are clearer, often sharp, valvular, or fœtal in character; whereas in effusion the sounds are distant and muffled.

(*e*) Rarely in dilatation is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region.

The number of excellent observers who have acknowledged that they have failed sometimes to discriminate between these two conditions, and who have indeed performed paracentesis *cordis* instead of paracentesis *pericardii*, is perhaps the best comment on the difficulties.

Massive (1½ to 2 litre) exudations have been confounded with a pleural effusion. On more than one occasion the pericardium has been tapped under the impression that the exudate was pleuritic. The flat tympany in the infrascapular region, the absence of well-defined movable dulness, and the feeble, muffled sounds are indicative points. If the case has been followed from day to day there is rarely much difficulty; but it is different when a case presents a large area of dulness in the antero-lateral region of the left chest, and there is no to-and-fro pericardial friction murmur. Many of the cases have been regarded as encapsulated pleural effusions.

The nature of the fluid cannot positively be determined without aspiration; but a fairly accurate opinion can be formed from the nature of the primary disease and the general condition of the patient. In rheumatic cases the exudation is usually sero-fibrinous; in septic and tuberculous cases it is often purulent from the outset; in senile, nephritic, and tuberculous cases the exudation is sometimes hæmorrhagic.

Treatment.—The patient should have absolute quiet, mentally and bodily, so as to reduce to a minimum the heart's action. Drugs given for this purpose, such as aconite or digitalis, are of doubtful utility. Local bloodletting by cupping or leeches is certainly advantageous in robust subjects, particularly in the cases of extension in pleuro-pneumonia. The ice-bag is of great value. It may be applied to the præcordia at first for an hour or more at a time, and then continuously. It reduces the frequency of the heart's action and seems to retard the progress of an effusion. Blisters are not indicated in the early stage.

When effusion is present, the following measures to promote absorption may be adopted: Blisters to the præcordia, a practice not so much in vogue now as formerly. It is surprising, however, in some instances, how quickly an effusion will subside on their application. If the patient's strength is good, a purge every other morning may be given. The diet should be light, dry, and nutritious. In cases in which the pulse is strong and the constitutional disturbance not great, iodide of potassium may be of service, and the action of the kidneys may be promoted by the infusion of digitalis and acetate of potash.

When the effusion is large, as soon as signs of serious impairment of the heart occur, as indicated by dyspnœa, small rapid pulse, dusky, anxious countenance, surgical measures should be resorted to, and paracentesis, or incision of the pericardium, at once be performed. With the sero-fibrinous exudate, such as commonly occurs after rheumatism, aspiration is sufficient; but when the exudate is purulent, the pericardium should be freely incised and freely drained. The puncture may be made in the fourth interspace, either at the left sternal margin or 2.5 cm. (an inch) from it. If made in the fifth interspace it is well to puncture an inch and a half from the left sternal margin. In large effusions the pericardium can also be readily reached without danger by thrusting the needle upward and back-

ward close to the costal margin in the left costo-xiphoid angle. The results of paracentesis of the pericardium have so far not been satisfactory. With an earlier operation in many instances and a more radical one in others—a free incision and not aspiration when the fluid is purulent—the percentage of recoveries will be greatly increased. Of 35 cases of suppurative pericarditis treated by incision 15 recovered and 20 died (Roberts, Am. Jr. Med. Sciences, Dec., 1897).

Chronic Adhesive Pericarditis (*Adherent Pericardium*).—Two groups of cases may be recognized:

(*a*) Simple adhesion of the peri- and epicardial layers. This is a common sequence of pericarditis, and is frequently met with post mortem as an accidental lesion. It is not necessarily associated with disturbance in the function of the heart, and in a large proportion of the cases there is neither dilatation nor hypertrophy.

(*b*) Adherent pericardium with chronic mediastinitis and union of the outer layer of the pericardium to the pleura and to the chest walls. This constitutes one of the most serious forms of cardiac disease, particularly in early life, and may lead to an extreme grade of hypertrophy and dilatation of the heart. Even with partial adhesion between the epicardium and pericardium there may be enormous hypertrophy under the conditions just mentioned. The *symptoms* of adherent pericardium are uncertain and indefinite. In the second group the features are those of hypertrophy and dilatation of the heart, later cardiac insufficiency, and in a few instances signs of extension of the mediastinitis to the peritonæum, causing chronic proliferative peritonitis, with perihepatitis and perisplenitis.* Sudden death may occur after an unusual exertion or during parturition (Reynolds Wilson).

The following are important points in the diagnosis: *Inspection.*—A majority of the signs of value come under this heading. (*a*) The præcordia is prominent and there may be marked asymmetry, owing to the enormous enlargement of the heart. (*b*) The extent of the cardiac impulse is greatly increased, and may sometimes be seen from the third to the sixth interspaces, and in extreme cases from the right parasternal line to outside the left nipple. (*c*) The character of the cardiac impulse. It is undulatory, wavy, and in the apex region there is marked systolic retraction. (*d*) Diaphragm phenomena. J. W. Broadbent has called attention to a very valuable sign in adherent pericardium. When the heart is adherent over a large area of the diaphragm there is with each pulsation a systolic tug, which may be communicated through the diaphragm to the points of its attachment on the wall, causing a visible systolic tugging. This has long been recognized in the region of the seventh or eighth ribs in the left parasternal line, but Dr. Broadbent called attention to the fact that it was frequently best seen on the left side behind, between the eleventh and twelfth ribs. With each systole there may be here a distinct, visible retraction of the chest wall. This is a very valuable and quite common sign. Sir William Broadbent calls attention also to the fact that owing to the attachment of the

* For illustrative cases see Arch. of Pediatrics, 1896.

heart to the central tendon of the diaphragm this part does not descend with inspiration, during which act there is not the visible movement in the epigastrium. (*e*) Diastolic collapse of the cervical veins, the so-called Friedreich's sign. This is not of much moment.

Palpation.—The apex beat is fixed, and turning the patient on the left side does not alter its position. This I have found, however, somewhat uncertain. On placing the hand over the heart there is felt a diastolic shock or rebound, which some have regarded as the most reliable of all signs of adherent pericardium.

Percussion.—The area of cardiac dulness is usually much increased. In a majority of instances there are adhesions between the pleura and the pericardium, and the limit of cardiac dulness above and to the left may be fixed and is uninfluenced by deep inspiration. This, too, is an uncertain sign, inasmuch as there may be close adhesions between the pleura and the pericardium and between the pleura and the chest wall, which at the same time allow a very considerable degree of mobility to the edge of the lung.

Auscultation.—The phenomena are variable and uncertain. In the cases in children with a history of rheumatism, endocarditis has usually been present. Even in the absence of chronic endocarditis, when the dilatation reaches a certain grade there are murmurs of relative insufficiency, which, as in one case I have recorded, may be present not only at the mitral but also at the tricuspid and pulmonary orifices. Hale White has called attention to the fact that there may be a well-marked presystolic murmur in connection with adherent pericardium. This was present in one of my cases.

The pulsus paradoxus, in which during inspiration the pulse-wave is small and feeble, is sometimes present, but it is not a diagnostic sign of either simple pericardial adhesion or of the cicatricial mediastino-pericarditis.

In children, chronic adhesive pericarditis and mediastinitis may be associated with proliferative peritonitis, perihepatitis, and perisplenitis, in which condition ascites may recur for months, or even for years.

II. OTHER AFFECTIONS OF THE PERICARDIUM.

(1) **Hydropericardium.**—Naturally there are in the pericardial sac a few cubic centimetres of clear, citron-colored fluid, which probably represents a post-mortem transudate. In certain conditions during life there may be a large secretion of serum forming what is known as dropsy of the pericardium. It occurs usually in connection with general dropsy, due to kidney or heart disease; more commonly the former. It rarely of itself proves fatal, though when the effusion is excessive it adds to the embarrassment of the heart and the lungs, particularly when the pleural cavities are the seat of similar exudation. There are rare instances in which effusion into the pericardium occurs after scarlet fever with few, if any, other dropsical symptoms. The physical signs are those already referred to in connection with pericarditis with effusion. It is frequently overlooked.

In rare cases the serum has a milky character—chylo-pericardium.

(2) **Hæmo-pericardium.**—This condition, by no means uncommon, is met with in aneurism of the first part of the aorta, of the cardiac wall, or of the coronary arteries, and in rupture and wounds of the heart. Death usually follows before there is time for the production of symptoms other than those of rapid heart-failure due to compression. Particularly is this the case in aneurism. In rupture of the heart the patient may live for many hours or even days with symptoms of progressive heart-failure, dyspnœa, and the physical signs of effusion.

As already mentioned, the inflammatory exudate of tubercle or cancer is often blood-stained. The same is true of the effusion in the pericarditis of Bright's disease and of old people.

(3) **Pneumo-pericardium.**—Gas is rarely found in the pericardial sac, and is due, as a rule, to perforation from without, as in the case of stab wounds, or is the result of perforation from the lungs, œsophagus, or stomach. Perforation from a tuberculous cavity is a not uncommon cause. In those cases, formerly so puzzling, in which the gas is present shortly after death (a few hours), the gas bacillus (*B. aërogenes capsulatus*) will be found. In a case at the Royal Victoria Hospital, in which the gas bacillus was isolated, the diagnosis was made during life (Nicholls). As a result of perforation, acute pericarditis is always excited, and the effusion rapidly becomes purulent. The physical signs are remarkable. When the effusion is copious the fluid and gas together give a movable area of percussion dulness with marked tympany in the region of the gas. On auscultation, remarkable splashing, churning, metallic phenomena are heard with friction and possibly feeble, distant heart-sounds. Death follows rapidly, even in thirty-six hours, as in a case (the only one which I have seen) of perforation of the pericardium in cancer of the stomach. Except as a result of injury, the condition is not one for which treatment is available. In a case of perforation from without with signs of effusion, to enlarge the wound by free incision would be justifiable.

II. DISEASES OF THE HEART.

I. ENDOCARDITIS.

Inflammation of the lining membrane of the heart is usually confined to the valves, so that the term is practically synonymous with valvular endocarditis. It occurs in two forms—*acute*, characterized by the presence of vegetations with loss of continuity or of substance in the valve tissues; *chronic*, a slow sclerotic change, resulting in thickening, puckering, and deformity.

Acute Endocarditis.

This occurs in rare instances as a primary, independent affection; but in the great majority of cases it is an accident in various infective processes, so that in reality the disease does not constitute an etiological entity.

A remarkable instance of strangulated hernia in the pericardium through an opening in the diaphragm the size of a sixpence has been reported by L. H. McDowell. Eighteen inches of strangulated ileum were in the pericardium. The symptoms were gastric - and he died suddenly. (B. M. J. p. 716, Vol. I-1897)

For convenience of description we speak of a simple or benign, and a malignant or ulcerative endocarditis, between which, however, there is no essential anatomical difference, as all gradations can be traced, and they represent but different degrees of intensity of the same process.

Etiology.—*Simple endocarditis* does not constitute a disease of itself, but is invariably found with some other affection. The general experience of the profession has confirmed the original observation of Bouillaud as to the frequency of association of simple endocarditis with acute articular rheumatism. Possibly it is nothing in the disease itself, but simply an altered state of the fluid media—a reduction perhaps of the lethal influences which they normally exert—permitting the invasion of the blood by certain micro-organisms. Tonsillitis, which in some forms is regarded as a rheumatic affection, may be complicated with endocarditis. Of the specific diseases of childhood it is not uncommon in scarlet fever, while it is rare in measles and chicken-pox. In diphtheria simple endocarditis is rare. In small-pox it is not common. In typhoid fever I have met with it twice in 80 autopsies.

In pneumonia both simple and malignant endocarditis are common. In 100 autopsies in this disease made at the Montreal General Hospital there were 5 instances of the former. Acute endocarditis is by no means rare in phthisis. I have met with it in 12 cases in 216 post mortems.

In chorea simple warty vegetations are found on the valves in a large majority of all fatal cases, in 62 of 73 cases collected by me. There is no disease in which, post mortem, acute endocarditis has been so frequently found. And, lastly, simple endocarditis is met with in diseases associated with loss of flesh and progressive debility, as cancer, and such disorders as gout, diabetes, and Bright's disease.

A very common form is that which occurs on the sclerotic valves in old heart-disease—the so-called recurring endocarditis.

Malignant endocarditis is met with: (*a*) As a primary disease of the lining membrane of the heart or of its valves.

(*b*) As a secondary affection in acute rheumatism, pneumonia, and in various specific fevers; or as an associated condition in septic processes.

It is also known by the names of ulcerative, infectious, or diphtheritic endocarditis, but the term malignant seems most appropriate to characterize the essential clinical features of the disease.

The existence of a primary endocarditis has been doubted; but there are instances in which persons previously in good health, without any history of affections with which endocarditis is usually associated, have been attacked with symptoms resembling severe typhus or typhoid. In one case which I saw, death occurred on the sixth day and no lesions were found other than those of malignant endocarditis.

The simple endocarditis of rheumatism rarely develops into the malignant form. In only 24 of 209 cases the symptoms of severe endocarditis arose in the progress of acute or subacute rheumatism. In only 3 of my Montreal cases was there a history of rheumatism either before or during the attacks.

Malignant endocarditis is extremely rare in chorea. Of all acute dis-

eases complicated with severe endocarditis pneumonia probably heads the list. This fact, which had been referred to by several of the older writers, was brought out in a striking manner by the figures on which my Gulstonian lectures were based. In 11 of the 23 Montreal cases the disease came on with lobar pneumonia, while it developed with this disease in 54 of the 209 cases analyzed—indeed, the endocarditis which occurs in pneumonia seems to be of an unusually malignant type, as in 16 cases of my 100 autopsies in this disease in which this lesion was present, 11 were of this form. This has been confirmed by Netter, Kanthack, and others. Meningitis was associated with endocarditis in 25 of the 209 cases, and in 15 there was also pneumonia.

The affection may complicate erysipelas, septicæmia (from whatever cause) and puerperal fever and gonorrhœa. Malignant endocarditis is very rare in tuberculosis, typhoid fever, and diphtheria.

It has been stated by many writers that endocarditis occurs in ague. With the unusual facilities for the study of this disease which I have had in the past nine years I have not yet met with an instance. Unquestionably, in the majority of these cases, the intermittent pyrexia, which has been regarded as characteristic of the ague, has depended upon the endocarditis. In dysentery cases have been described. In small-pox and scarlet fever, with which simple endocarditis is not infrequently complicated, the malignant form is extremely rare.

Morbid Anatomy of Simple and Malignant Endocarditis.—*Simple endocarditis* is characterized by the presence on the valves or on the lining membrane of the chambers of minute vegetations, ranging from 1 to 4 mm. in diameter, with an irregular and fissured surface, giving to them a warty or verrucose appearance. Often these little cauliflower-like excrescences are attached by very narrow pedicles. They are more common on the left side of the heart than the right, and occur on the mitral valves more often than on the aortic. The vegetations are usually above the line of closure of the valves. It is rare to see any swelling or macroscopic evidence of infiltration of the endocardium in the neighborhood of even the smallest of the granulations, and redness, indicative of distention of the vessels, is uncommon, even when they occur upon valves already the seat of sclerotic changes, in which capillary vessels extend to the edges. With time the vegetations may increase greatly in size, but in what may be called simple endocarditis the size rarely exceeds that mentioned above.

The earliest vegetations consist of elements derived from the blood, and are composed of blood platelets, leucocytes, and fibrin in varying proportions. At a later stage they appear as small outgrowths of connective tissue. The transition of one form into the other can often be followed. The process consists of a proliferation of the endothelial cells and the cells of the subendothelial layer which gradually invade the fresh vegetation, and ultimately entirely replace it. The blood-cells and fibrin undergo disintegration and gradually they are removed. The whole process has received the name of "organization." Even when the vegetation has been entirely converted into granulations or connective tissue it is often found at autopsy to be capped with a thin layer of fibrin and leucocytes.

Micro-organisms are generally, even if not invariably, found associated with the vegetations. They tend to be entangled in the granular and fibrillated fibrin or in the older ones to cap the apices.

In both man and animals there is a form of *chronic vegetative endocarditis* in which, without much or any loss of substance, the valves and chordæ tendineæ are covered with large, firm outgrowths. In several cases of this kind the clinical history has been characterized by a protracted fever of a marked remittent or even intermittent type.

Subsequent Changes.—(1) The vegetations may become organized and the valve restored to a normal state (?). (2) The process may extend, and a simple may become an ulcerative endocarditis. (3) The vegetations may be broken off and carried in the circulation to distant parts. (4) The vegetations become organized and disappear, but they initiate a nutritive change in the valve tissue which ultimately leads to sclerosis, thickening, and deformity. The danger in any case of simple endocarditis is not immediate, but remote, and consists in this perversion of the normal processes of nutrition which results in sclerosis of the valves.

A gradual transition from the simple to a more severe affection, to which the name *malignant* or *ulcerative endocarditis* has been given, may be traced. Practically every case of ulcerative endocarditis is attended by vegetations. In this form the loss of substance in the valve is more pronounced, the deposition—thrombus formation—from the blood is more extensive, and the micro-organisms are present in greater number and often show increased virulence. Ulcerative endocarditis is often found in connection with heart valves already the seat of chronic proliferative and sclerotic changes.

In malignant endocarditis there is distinct loss of substance in the heart valve. This loss may be superficial and limited to the endocardium, or, what is more common, it involves deeper structures, and not very infrequently leads to perforation of a valve, a septum, or even of the heart itself.

Upon microscopical examination the affected valve shows necrosis, with more or less loss of substance; the necrotic tissue is devoid of preserved nuclei and presents a coagulated appearance. Upon it a mixture of blood platelets, fibrin—granular or fibrillated—and leucocytes enclosing masses of micro-organisms are met with. The subjacent tissue often shows sclerotic thickening and always infiltration with exuded granulation tissue-cells.

Parts affected.—The following figures, taken from my Gulstonian lectures at the Royal College of Physicians, give an approximate estimate of the frequency with which in 209 cases different parts of the heart were affected in malignant endocarditis: Aortic and mitral valves together, in 41; aortic valves alone, in 53; mitral valves alone, in 77; tricuspid in 19; the pulmonary valves in 15; and the heart walls in 33. In 9 instances the right heart alone was involved, in most cases the auriculo-ventricular valves.

Mural endocarditis is seen most often at the upper part of the septum of the left ventricle. Next in order is the endocarditis of the left auricle on the postero-external wall. The vegetations may extend, as in a recent case in my wards, along the intima of the pulmonary artery into the hilum of the lung. The ulcerative changes may lead to perforation of a valve segment, erosion of the chordæ tendineæ, perforation of the septum, or even

of the heart itself. A common result of the ulceration is the production of valvular aneurism. In three fourths of the cases the affected valves present old sclerotic changes. The process may extend to the aorta, producing, as in one of my cases, extensive endarteritis with multiple acute aneurisms.

Associated Lesions.—The associated pathological changes are partly those of the primary disease to which the endocarditis is secondary and partly those due to embolism. In the endocarditis of septic processes there is the local lesion—an acute necrosis, a suppurative wound, or puerperal disease. In many cases the lesions are those of pneumonia, rheumatism, or other febrile processes. The changes due to embolism constitute the most striking features, but it is remarkable that in some instances, even with endocarditis of a markedly ulcerative character, there may be no trace of embolic processes.

The infarcts may be few in number—only one or two, perhaps, in the spleen or kidney—or they may exist in hundreds throughout the various parts of the body. They may present the ordinary appearance of red or white infarcts of a suppurative character. They are most common in the spleen and kidneys, though they may be numerous in the brain, and in many cases are very abundant in the intestines. In right-sided endocarditis there may be infarcts in the lungs. In many of the cases there are innumerable miliary abscesses. Acute suppurative meningitis was met with in 5 of 23 of the Montreal cases, and in over 10 per cent of the 209 cases analyzed in the literature. Acute suppurative parotitis also may occur.

Bacteriology.—No distinction in the micro-organisms found in the two forms of endocarditis can be made. In both the pyogenic cocci—streptococci, staphylococci, pneumococci, and gonococci—are the most frequent bacteria met with. More rarely, especially in the simple vegetative endocarditis, the bacilli of tuberculosis, typhoid fever, and anthrax have been encountered. The bacillus coli communis has also been found, and Howard has described a case of malignant endocarditis due to an attenuated form of the diphtheria bacillus. Flexner * has analyzed 34 cases of acute endocarditis associated with chronic renal and cardiac disease, and found the micrococcus lanceolatus and the streptococcus pyogenes present each twelve times, the staphylococcus three times. Other bacteria encountered were bacillus pyocyaneus, coli, and influenzæ, and the gonococcus.

Symptoms.—Neither the clinical course nor the physical signs of *simple endocarditis* are in any respect characteristic. The great majority of the cases are latent and there is no indication whatever of cardiac mischief. Experience has taught us that endocarditis is frequently found post mortem in persons in whom it was not suspected during life. There are certain features, however, by which its presence is indicated with a degree of probability. The patient, as a rule, does not complain of any pain or cardiac distress. In a case of acute rheumatism, for example, the symptoms to excite suspicion would be increased rapidity of the heart's action, perhaps slight irregularity, and an increase in the fever without aggravation

* Journal of Experimental Medicine, 1896, i, p. 559.

of the joint trouble. Rows of tiny vegetations on the mitral or on the aortic segments seem a trifling matter to excite fever, and it is difficult in the endocarditis of febrile processes to say definitely in every instance that an increase in the fever depends upon the endocardial complication. But a study of the recurring endocarditis—which is of the warty variety, consisting of minute beads on old sclerotic valves—shows that this process may be associated, for days or weeks at a time, with slight fever ranging from 100° to 102½°. Palpitation may be a marked feature and is a symptom upon which certain authors lay great stress.

The *diagnosis* of the condition rests upon physical signs which are notoriously uncertain. The presence of a murmur at one or other of the cardiac areas in a case of fever is often regarded as indicative of the existence of endocarditis. This extremely common mistake has arisen from the fact that the *bruit de souffle* or bellows murmur is common to endocarditis and a number of other conditions which have nothing to do with it. At first there may be only a slight roughening of the first sound, which may gradually develop into a distinct murmur. Taken alone, it is, however, a very uncertain and fallacious sign.

It is difficult to give a satisfactory clinical picture of *malignant endocarditis* because the modes of onset are so varied and the symptoms so diverse. Arising in the course of some other disease, there may be simply an intensification of the fever or a change in its character. In a majority of the cases there are present certain general features, such as irregular pyrexia, sweating, delirium, and gradual failure of strength.

Embolic processes may give special characters, such as delirium, coma or paralysis from involvement of the brain or its membranes, pain in the side and local peritonitis from infarction of the spleen, bloody urine from implication of the kidneys, impaired vision from retinal hæmorrhage, and suppuration, and even gangrene, in various parts from the distribution of the emboli.

Two special types of the disease have been recognized—the septic or pyæmic and the typhoid. Other cases closely resemble true intermittent fever. In some the cardiac symptoms are most prominent, while in others again the main symptoms may be those of an acute affection of the cerebro-spinal system.

The *septic type* is met with usually in connection with an external wound, the puerperal process, or an acute necrosis. There are rigors, sweats, irregular fevers, and all of the signs of septic infection. The heart symptoms may be completely masked by the general condition, and attention called to them only on the occurrence of embolism. In a most remarkable sub-group of this type the disease may simulate a quotidian or a tertian ague. The symptoms may develop in persons with chronic heart-disease without any external lesions. These cases may be much prolonged—for three or four months, or even longer, as in one of Bristowe's. The existence in some of these instances of a previous genuine malaria has been a very puzzling circumstance.

The *typhoid type* is by far the most common and is characterized by an irregular temperature, early prostration, delirium, somnolence, and coma,

relaxed bowels, sweating, which may be of a most drenching character, petechial and other rashes, and occasionally parotitis. The heart symptoms may be completely overlooked, and in some instances the most careful examination has failed to discover a murmur.

Under the *cardiac group*, as suggested by Bramwell, may be considered those cases in which patients with chronic valve disease are attacked with marked fever and evidence of recent endocarditis. Many such cases present symptoms of the pyæmic and typhoid character and may run a most acute course. In others the course is chronic, lasting for weeks or months. I have reported two cases of this chronic vegetative endocarditis, with intermittent fever, one of more than a year's duration. The autopsies showed extensive vegetative and ulcerative disease of the mitral valves.

There are cases in which it is often difficult to decide whether malignant endocarditis is present or not. Thus, a patient with aortic valve disease is under treatment for failing compensation and begins to have irregular fever with restlessness and cardiac distress; embolic phenomena may develop—sudden hemiplegia, pain in the region of the spleen, or bloody urine, or perhaps peripheral embolism. There may be a low delirium and the case may run a tolerably acute course; but in other instances the fever subsides and recovery occurs.

In what may be termed the *cerebral group* of cases the clinical picture may simulate a meningitis, either basilar or cerebro-spinal. There may be acute delirium or, as in three of the Montreal cases, the patient may be brought into the hospital unconscious. Heineman reports an instance, with autopsy, in which the clinical picture was that of an acute cerebro-spinal meningitis.

Certain special symptoms may be mentioned. The fever is not always of a remittent type, but may be high and continuous. Petechial rashes are very common and render the similarity very strong to certain cases of typhoid and cerebro-spinal fever. In one case the disease was thought to be hæmorrhagic small-pox. Erythematous rashes are not uncommon. The sweating may be most profuse, even exceeding that which occurs in phthisis and ague. Diarrhœa is not necessarily associated with embolic lesions in the intestines. Jaundice has been observed and cases are on record which were mistaken for acute yellow atrophy.

The heart symptoms may be entirely latent and are not found unless a careful search be made. Even on examination there may be no murmur present. Instances are recorded by careful observers, in which the examination of the heart has been negative. Cases with chronic valve disease usually present no difficulty in diagnosis.

The course of the disease is varied, depending largely upon the nature of the primary trouble. Except in the disease grafted upon chronic valvulitis the course is rarely extended beyond five or six weeks. As already mentioned, there are instances in which the disease is prolonged for months. The most rapidly fatal case on record is described by Eberth, the duration of which was scarcely two days.

Diagnosis.—In many cases the detection of the disease is very difficult; in others, with marked embolic symptoms, it is easy. From simple

endocarditis it is readily distinguished, though confusion occasionally occurs in the transitional stage, when a simple is developing into a malignant form. The constitutional symptoms are of a graver type, the fever is higher, rigors are common, and septic and typhoid symptoms develop. Perhaps a majority of the cases not associated with puerperal processes or bone-disease are confounded with typhoid fever. A differential diagnosis may even be impossible, particularly when we consider that in typhoid fever infarctions and parotitis may occur. The diarrhœa and abdominal tenderness may also be present, which with the stupor and progressive asthenia make a picture not to be distinguished from this disease. Points which may guide us are: The more abrupt onset in endocarditis, the absence of any regularity of the pyrexia in the early stage of the disease, and the cardiac pain. Oppression and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. There is a marked leucocytosis in infective endocarditis. Between pyæmia and malignant endocarditis there are practically no differential features, for the disease really constitutes an *arterial pyæmia* (Wilks). In the acute cases resembling malignant fevers, the diagnosis is usually made of typhus, typhoid, cerebro-spinal fever, or even of hæmorrhagic small-pox. The intermittent pyrexia, occurring for weeks or months, has led in some cases to the diagnosis of malaria, but this disease could now be positively excluded by the blood examination.

The cases usually terminate fatally. The instances of recovery are those more subacute forms, the so-called recurring endocarditis developing on old sclerotic valves in cases of chronic heart-disease.

Treatment.—We know no measures by which in rheumatism, chorea, or the eruptive fevers the onset of endocarditis can be prevented. As it is probable that many cases develop, particularly in children, in mild forms of these diseases, it is well to guard the patients against taking cold and insist upon rest and quiet, and to bear in mind that of all complications an acute endocarditis, though in its immediate effects harmless, is perhaps the most serious. This statement is enforced by the observations of Sibson that on a system of absolute rest the proportion of cases of rheumatism attacked by endocarditis was less than of those who were not so treated.

It is doubtful whether the salicylates in rheumatism have an influence in reducing the liability to endocarditis. When the endocarditis is present we know no remedies which will definitely influence the valvular lesions. If there is much vascular excitement aconite may be given and an ice-bag placed over the heart.

The salicylates are strongly advised by some writers and the sulphocarbolates have been recommended by Sansom. In the severer cases of malignant endocarditis the treatment is practically that of septicæmia.

Chronic Endocarditis.

This condition, which is a sclerosis of the valve, may be primary, but is oftener secondary to acute endocarditis, particularly the rheumatic form.

It is essentially a slow, insidious process which leads to deformity of the valve segment and is the foundation of chronic valvular disease.

Certain poisons appear capable of initiating the change, such as alcohol, syphilis, and gout, though we are at present ignorant of the way in which they act. A very important factor, particularly in the case of the aortic valves, is the strain of prolonged and heavy muscular exertion. In no other way can be explained the occurrence of so many cases of sclerosis of the aortic valves in young and middle-aged men whose occupations necessitate the overuse of the muscles.

Morbid Anatomy.—Vegetations in the form in which they occur in acute endocarditis are not present. In the early stage, which we have frequent opportunities of seeing, the edge of the valve is a little thickened and perhaps presents a few small nodular prominences, which in some cases may represent the healed vegetations of the acute process. In the aortic valves the tissue about the corpora Arantii is first affected, producing a slight thickening with an increase in the size of the nodules. The substance of the valve may lose its translucency, and the only change noticeable be a grayish opacity and a slight loss of its delicate tenuity. In the auriculo-ventricular valves these early changes are seen just within the margin and here it is not uncommon to find swellings of a grayish-red, somewhat infiltrated appearance, almost identical with the similar structures on the intima of the aorta in arterio-sclerosis. Even early there may be seen yellow or opaque-white subintimal fattily degenerated areas. As the sclerotic changes increase, the fibrous tissue contracts and produces thickening and deformity of the segment, the edges of which become round, curled, and incapable of that delicate apposition necessary for perfect closure. A sigmoid valve, for instance, may be narrowed one fourth or even one third across its face, the most extreme grade of insufficiency being induced without any special deformity and without any definite narrowing of the arterial orifice. In the auriculo-ventricular segments a simple process of thickening and curling of the edges of the valves, inducing a failure to close without forming any obstruction to the normal course of the blood-flow, is less common. Still, we meet with instances at the mitral orifice, particularly in children, in which the edges of the valves are curled and thickened, so that there is extreme insufficiency without any material narrowing of the orifice. More frequently, as the disease advances, the chordæ tendineæ become thickened, first at the valvular ends and then along their course. The edges of the valves at their angles are gradually drawn together and there is a definite narrowing of the orifice, leading in the aorta to more or less stenosis and in the left auriculo-ventricular orifice—the two sites most frequently involved—to constriction. Finally, in the sclerotic and necrotic tissues lime salts are deposited and may even reach the deeper structures of the fibrous rings, so that the entire valve becomes a dense calcareous mass with scarcely a remnant of normal tissue. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valve. The apices of the papillary muscles usually show marked fibroid change.

In all stages of the process the vegetations of simple endocarditis may be present, and upon sclerotic valves we find the severer, ulcerative form of the disease.

Chronic *mural* endocarditis produces cicatricial-like patches of a grayish-white appearance which are sometimes seen on the muscular trabeculæ of the ventricle or in the auricles. It often occurs in association with myocarditis.

The frequency with which chronic endocarditis is met with may be gathered from the following figures: In the statistics, amounting to from 12,000 to 14,000 autopsies, reported from Dresden, Würzburg, and Prague the percentage ranged from four to nine. The relative frequency of involvement of the various valves is thus given in the collected statistics of Parrot: The mitral orifice was involved in 621, the aortic in 380, the tricuspid in 46, and the pulmonary in 11. This gives 57 instances in the right to 1,001 in the left heart.

The endocarditis of the fœtus is usually of the sclerotic form and involves the valves of the right more frequently than those of the left side.

II. CHRONIC VALVULAR DISEASE.

1. General Introduction.

The *incidence* of valvular lesions may be gathered from the following figures compiled by Gillespie from the records of the Royal Infirmary, Edinburgh: Of 2,368 cases with cardiac lesions, valvular disease occurred in 80.8 per cent; endocarditis and pericarditis in 5.3; myocardial lesions in 11.9 per cent; 66.2 per cent of the cases were in males.

Effects of Valve Lesions.—The general influence on the work of the heart may be briefly stated as follows: The sclerosis induces insufficiency or stenosis, which may exist separately or in combination. The narrowing retards in a measure the normal outflow and the insufficiency permits the blood current to take an abnormal course. In both instances the effect is dilatation of a chamber. The result in the former case is an increase in the difficulty which the chamber has in expelling its contents through the narrow orifice; in the other, the overfilling of a chamber by blood flowing into it from an improper source, as, for instance, in mitral insufficiency, when the left auricle receives blood both from the pulmonary veins and from the left ventricle.

The cardiac mechanism is fully prepared to meet ordinary grades of dilatation which constantly occur during sudden exertion. A man, for instance, at the end of a hundred-yard race has his right chambers greatly dilated and his reserve cardiac power worked to its full capacity. The slow progress of the sclerotic changes brings about a gradual, not an abrupt, insufficiency, and the moderate dilatation which follows is at first overcome by the exercise of the ordinary reserve strength of the heart muscle. Gradually a new factor is introduced. The reserve power which is capable of meeting sudden emergencies in such a remarkable manner is unable to cope

long with a permanent and perhaps increasing dilatation. More work has to be done and, in accordance with definite physiological laws, more power is given by increase of the muscles. The heart hypertrophies and the effect of the valve lesion becomes, as we say, *compensated*. The equilibrium of the circulation is in this way maintained.

The nature of the process with which we have to deal is graphically illustrated in the accompanying diagrams, which we owe to Martius, of Rostock. The perpendicular lines in the figures represent the power of work of the heart. While the muscle in the healthy heart (Diagram I) has at its disposal the maximal force, $a\,c$, it carries on its work under ordinary circumstances (when the body is at rest) with the force $a\,b$. The force $b\,c$ is reserve force, by means of which the heart accommodates itself to greater exertion.

If now there be a gross valvular lesion, the force required to do the ordinary work of the heart (at rest) becomes very much increased (Diagram II). But in spite of this enormous call for force, insufficiency of the heart muscle does not necessarily result, for the working force required is still within the

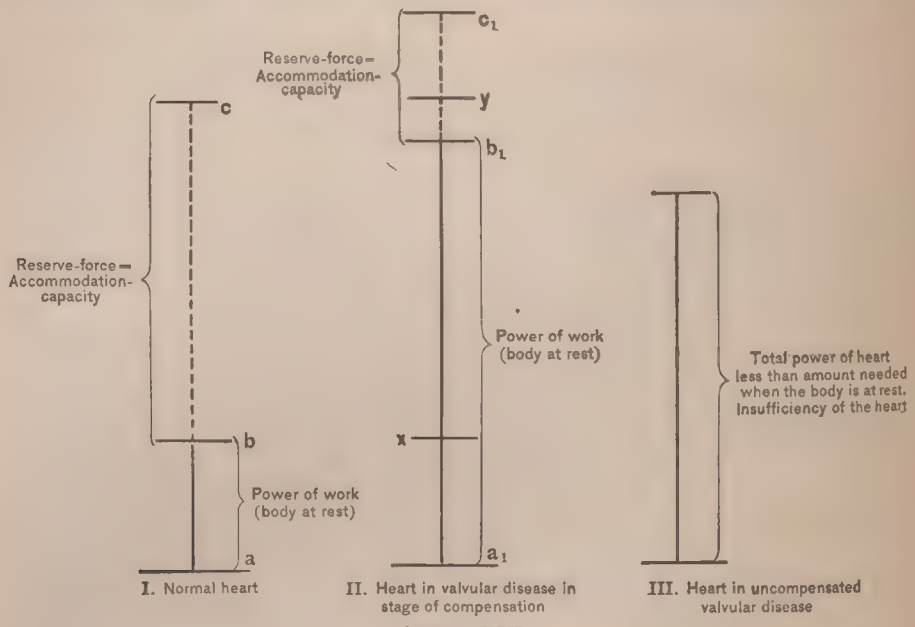

CHART XVI.

limits of the maximal power of the heart, $a_1\,b_1$, being less than $a_1\,c_1$. The muscle accommodates itself to the new conditions by making its reserve force mobile (experiment of Rosenbach). If nothing further occurred, however, this condition could not be permanently maintained, for there would be left over for emergencies only the small reserve force, $b_1\,y$. Even when at rest the heart would be using continuously almost its entire maximal force. Any slight exertion requiring more extra force than that represented by the small value $b_1\,y$ (say the effort required on walking or on

going upstairs) would bring the heart to the limit of its working power, and palpitation and dyspnœa would appear. Such a condition does not last long. The working power of the heart gradually increases. More and more exertion can be borne without causing dyspnœa, for *the heart hypertrophies*. Finally, a new, more or less permanent condition is attained, in that the hypertrophied heart possesses the maximal force, *a*, *c*. Owing to the increase in volume of the heart muscle, the total force of the heart is greater *absolutely* than that of the normal heart by the amount *y*, *c*. It is, however, *relatively* less efficient, for its reserve force is much less than that of the healthy heart. Its capacity for accommodating itself to unusual calls upon it is accordingly permanently diminished.

Turning now to the disturbances of compensation, it is to be distinctly borne in mind that any heart, normal or diseased, can become insufficient whenever a call upon it exceeds its maximal working capacity. The liability to such disturbance will depend, above all, upon the accommodation limits of the heart—the less the width of the latter, the easier will it be to go beyond the heart's efficiency. A comparison of Diagrams I and II will immediately make it clear that the heart in valvular disease will much earlier become insufficient than the heart of a healthy individual. If the heart muscle is compelled to do maximal or nearly maximal work for a long time, it becomes exhausted. It is obvious that the heart in valvular disease has on account of its small amount of reserve force to do maximal or nearly maximal work far more frequently than does the normal heart. The power of the heart may become decreased to the amount necessary simply to carry on the work of the heart when the body is at rest, or it may cease to be sufficient even for this. The reserve force gained through the compensatory process may be entirely lost (Diagram III). If the loss be only temporary, the exhausted heart muscle quickly recovering, the condition is spoken of as a "disturbance of compensation." The term "loss of compensation" is reserved for the condition in which the disturbance is continuous.

2. Aortic Incompetency.

Incompetency of the aortic valves arises either from inability of the valve segments to close an abnormally large orifice or more commonly from disease of the segments themselves. This best-defined and most easily recognized of valvular lesions was first carefully studied by Corrigan, whose name it sometimes bears.

Etiology and Morbid Anatomy.—It is more frequent in males than in females, affecting chiefly able-bodied, vigorous men at the middle period of life. The ratio which it bears to other valve diseases has been variously given from 30 to 50 per cent.

Among the important factors in producing this condition are: (*a*) Congenital malformation, particularly fusion of two segments—most commonly those behind which the coronary arteries are given off. It is probable that an aortic orifice may be competent with this bicuspid state of the valves, but a great danger is the liability of these malformed segments to sclerotic endocarditis. Of 17 cases which I have reported all presented

sclerotic changes, and the majority of them had, during life, the clinical features of chronic heart-disease.

(*b*) Acute endocarditis. This does not produce aortic incompetency unless the process passes on to ulceration and destruction, under which circumstances it is often found, and may cause a rapidly fatal issue. Simple endocarditis associated with the specific fevers is not nearly so common on the aortic as on the mitral segments; so also with rheumatism, which plays a less important *rôle* here than in mitral valve disease.

(*c*) By far the most frequent cause of insufficiency is a slow, progressive sclerosis of the segments, resulting in a curling of the edges, which lessens the working surface of the valve. This may, of course, follow acute endocarditis, but it is so often met with in strong, able-bodied men among the working classes, without any history of rheumatism or special febrile diseases with which endocarditis is commonly associated, that other conditions must be sought for to explain its frequency. Of these, unquestionably strain is the most important—not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle. Of circumstances increasing this tension, heavy and excessive use of the muscles is perhaps the most important. So often is this form of heart-disease found in persons devoted to athletics that it is sometimes called the "athlete's heart." Alcohol is a second important factor, and is stated to raise considerably the tension in the aortic system. A combination of these two causes is extremely common. A third element in inducing chronic sclerotic changes in these valves is syphilis. Cases are rarely seen in which other factors must not be taken into account, but the association is too frequent to be accidental. That syphilis is capable of inducing arterial sclerosis is, I think, acknowledged, although the way in which it does so is not yet clear. It is interesting to note with what frequency this form of valve disease occurs in soldiers. I was struck with this fact in the Philadelphia Hospital, to which so many veterans of the civil war are admitted. I was in the habit of enforcing upon my students the etiological lesson by a reference to Bacchus and Vulcan, at whose shrines a majority of the cases of aortic insufficiency have worshipped, and not a few at those of Mars and Venus.

The condition of the valves is such as has already been described in chronic endocarditis. It may be noted, however, how slight a grade of curling may produce serious incompetency. Associated with the valve disease is, in a majority of the cases, a more or less advanced arterio-sclerosis of the arch of the aorta, one serious effect of which may be a narrowing of the orifices of the coronary arteries. The sclerotic changes are often combined with atheroma, either in the fatty or calcareous stage. This may exist at the attached margin of the valves without inducing insufficiency. In other instances insufficiency may result from a calcified spike projecting from the aortic attachment into the body of the valve, and so preventing its proper closure. Some writers (Peter) have laid great stress upon the extension of the endarteritis to the valve, and would separate the instances of this kind from those of simple valvular endocarditis. I must say that I have not been able to recognize clinical differences between these two con-

ditions, though anatomically we may separate the cases into two groups — the endocarditic and the arterio-sclerotic.

(*d*) And, lastly, insufficiency may be induced by rupture of a segment —a very rare event in healthy valves, but not uncommon in disease, either from excessive strain during heavy lifting or from the ordinary endarterial strain in a valve eroded and weakened by ulcerative endocarditis.

Relative insufficiency of the sigmoid valves, due to dilatation of the aortic ring, is a rare condition. It is said to occur in extensive arterial sclerosis of the ascending portion of the arch with great dilatation just above the valves. In such cases the valve segments are usually involved with the arterial coats. In aneurism just above the aortic ring, relative insufficiency of the valve may be present.

It would appear from the careful measurements of Bencke that the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one-and-twenty it is about 60 mm. At this it remains until the age of forty, beyond which date there is a gradual increase in the size up to the age of eighty, when it may reach from 68 to 70 mm. There is thus at the very period of life in which sclerosis of the valve is most common a physiological tendency toward the production of a state of relative insufficiency.

The insufficiency may be combined with various grades of narrowing, but the majority of the cases of aortic insufficiency present no signs of stenosis. On the other hand, cases of aortic stenosis almost without exception are associated with some grade, however slight, of regurgitation.

The direct effect of aortic insufficiency is the regurgitation of blood from the artery into the ventricle, causing an overdistention of the cavity and a reduction of the blood column; that is, a relative anæmia in the arterial tree. As an immediate effect of the double blood-flow into the left ventricle dilatation of the chamber occurs, and finally hypertrophy. In this way the valve defect is compensated and as with each ventricular systole a larger amount of blood is propelled into the arterial system, the regurgitation of a certain amount during diastole does not, for a time at least, seriously impair the nutrition of the peripheral parts. In this valve lesion dilatation and hypertrophy reach their most extreme limit. The heaviest hearts on record are described in connection with this affection. The so-called bovine heart, *cor bovinum*, may weigh 35 or 40 ounces, or even, as in a case of Dulles's, 48 ounces. The dilatation is usually extreme, and is in marked contrast to the condition of the chamber in cases of pure aortic stenosis. The papillary muscles may be greatly flattened. The mitral valves are usually not seriously affected, though the edges may present slight sclerosis, and there is often relative incompetency, owing to distention of the mitral ring. Dilatation and hypertrophy of the left auricle are common, and secondary enlargement of the right heart occurs in all cases of long standing. The myocardium usually presents changes, fibroid or fatty; more commonly the former in association with disease of the coronary arteries. The arch of the aorta may present extensive arterio-sclerosis and dilatation. In the endocarditic cases, particularly those following rheumatism, the intima is perfectly smooth, and the arch with its main branches

not dilated. This condition may be found post mortem even when during life there have been the most characteristic signs of enlargement of the arch and of dilatation of the innominate and right carotid. I have even known the condition of aneurism to be diagnosed when post mortem no trace of dilatation or sclerosis was found, only an extreme grade of insufficiency with enormous dilatation and hypertrophy. The coronary arteries are usually involved in the sclerosis, and their orifices may be much narrowed. Although these vessels have been shown by Martin and Sedgwick to be filled during the ventricular systole, the circulation in them must be embarrassed in aortic incompetency. They must miss the effect of the blood-pressure in the sinuses of Valsalva during the elastic recoil of the arteries, which surely aids in keeping the coronary vessels full. The arteries of the body usually present more or less sclerosis consequent upon the strain which they undergo during the forcible ventricular systole.

Symptoms.—The condition is often discovered accidentally in persons who have not presented any features of cardiac disease.

Headache, dizziness, flashes of light, and a feeling of faintness on rising quickly are among the earliest symptoms. Palpitation and cardiac distress on slight exertion are common. Long before any signs of failing compensation pain may become a marked and troublesome feature. It is extremely variable in its manifestations. It may be of a dull, aching character confined to the præcordia. More frequently, however, it is sharp and radiating, and is transmitted up the neck and down the arms, particularly the left. Attacks of true angina pectoris are more frequent in this than in any other valvular disease. Anæmia is also common, much more so than in aortic stenosis or in mitral affections.

More serious symptoms, as compensation fails, are shortness of breath and œdema of the feet. The attacks of dyspnœa are liable to come on at night, and the patient has to sleep with the head high or even in a chair. Cyanosis is rare. It is most commonly due to complicating valve disease, or it is stated that it may result from bulging of the septum ventriculorum and encroachment upon the right ventricle. Of respiratory symptoms cough may develop, due to the congestion of the lungs or œdema. Hæmoptysis is less frequent than in mitral disease. I have reported a case in which it was profuse and believed to be due to tuberculosis of the lungs, inasmuch as the patient was admitted in a state of emaciation and profound exhaustion. General dropsy is not common, but œdema of the feet may occur early and is sometimes due to the anæmia, at others to the venous stasis, at times to both. Unless there is coexisting disease of the mitral valve, it is rare in aortic incompetency for the patient to die with general anasarca. Sudden death is frequent; more so in this than in other valvular diseases. As compensation fails the patient takes to bed and slight irregular fever, associated usually with a recurring endocarditis, is not uncommon toward the close. Embolic symptoms are not infrequent—pain in the splenic region with enlargement of the organ, hæmaturia, and in some cases paralysis. Distressing dreams and disturbed sleep are more common in this than in other forms of valvular disease.

Here may appropriately be mentioned the connection between mental

symptoms and cardiac disease, as they are oftenest seen with this lesion. An admirable account of the relations between insanity and disease of the heart is to be found in Mickle's Gulstonian lectures for 1888. In general medical practice we seldom find marked mental symptoms, except toward the close of the disease, when there may be delirium, hallucinations, and morbid impulses. It is to be remembered that in many heart cases this terminal delirium is uræmic. The irritability and peevishness sometimes found in persons the subject of organic heart-disease cannot, I think, be associated with it in any special manner. We do meet insanity, breaking out in patients with aortic and mitral disease, in the stage of compensation, which appears to be related definitely to the cardiac lesion. It is important to bear this in mind, for cases occasionally display suicidal tendencies. I have twice had patients throw themselves from a window of the ward.

Physical Signs.—*Inspection* shows a wide and forcible area of cardiac impulse with the apex beat in the sixth or seventh interspace, and perhaps as far out as the anterior axillary line. In young subjects the præcordia may bulge. On palpation a thrill, diastolic in time, is occasionally felt, but is not common. The impulse is usually strong and heaving, unless in conditions of extreme dilatation, when it is wavy and indefinite. Occasionally two or three interspaces between the nipple line and sternum will be depressed with the systole as a result of atmospheric pressure. *Percussion* shows a greater increase in the area of heart dulness than is found in any other valvular lesion. It extends chiefly downward and to the left.

On *auscultation* there is heard a murmur during diastole in the second right interspace, which is propagated with intensity toward the ensiform cartilage, or down the left margin of the sternum toward the apex. In the majority of cases it is a soft, long-drawn *bruit*, and is of all cardiac murmurs the most trustworthy. It occurs during the time of, and is produced by, the reflux of blood from the aorta into the ventricle. In a large proportion of the cases there is also a systolic murmur heard at the aortic region, usually shorter, often rougher in quality, and which may be propagated upward into the neck. A common mistake is to regard this as indicating stenosis, whereas in the great majority of instances of aortic insufficiency there is no material narrowing, and the murmur is produced by roughening of the segments or of the intima of the arch. The second sound is usually obliterated, but when the valves are only slightly curled or if one cusp only is involved both the murmur and the valvular sound may be distinctly heard. At the apex murmurs are also heard, either transmitted from the aortic orifice or produced at the mitral. In the majority of cases with aortic incompetency of high grade, the mitral orifice is dilated, and there is relative insufficiency of the valves. It can frequently be determined that the systolic murmur at the apex differs in quality from that at the base. A second murmur at the apex, probably produced at the mitral orifice, is not uncommon. Attention was called to this by the late Austin Flint, and the murmur usually goes by his name. It has a distinctly rumbling quality, is limited in area, and is sometimes, though not always, exactly presystolic in time. The explanation of its occurrence, as given by Flint, is that in the extreme dilatation of the ventricle the mitral segments

cannot during diastole be forced back against the wall, and therefore, remaining in the blood current, they produce a sort of relative narrowing, and in consequence a vibratory murmur not unlike in quality the presystolic murmur of mitral stenosis. Broadbent, on the other hand, suggests that the regurgitant current from the aorta impinging upon the anterior or aortic flap of the mitral may set it into vibration and thus produce the murmur. This apex diastolic murmur of aortic insufficiency occurs in a considerable proportion of all cases. It is variable, and may disappear as the dilatation of the ventricle diminishes. There is never the loud systolic shock which follows the murmur of mitral stenosis.

The examination of the arteries in aortic insufficiency is of great value. Visible pulsation is more commonly seen in the peripheral vessels in this than in any other condition. The carotids may be seen to throb forcibly, the temporals to dilate, and the brachials and radials to expand with each heart-beat. With the ophthalmoscope the retinal arteries are seen to pulsate. Not only is the pulsation evident, but the characteristic jerking quality is apparent. In the throat the throbbing carotids may lead to the diagnosis of aneurism. In many cases the pulsation can be seen in the suprasternal notch, and prominent, forcibly-throbbing vessels beneath the right sterno-mastoid muscle. The abdominal aorta may lift the epigastrium with each systole. To be mentioned with this is the capillary pulse, met very often in aortic insufficiency, and best seen in the finger-nails or by drawing a line upon the forehead, when the margin of hyperæmia on either side alternately blushes and pales. In extreme grades the face or the hand may blush visibly at each systole. It is met with also in profound anæmia, occasionally in neurasthenia, and in health in conditions of great relaxation of the peripheral arteries. Pulsation may also be present in the peripheral veins. On palpation the characteristic water-hammer or Corrigan pulse is felt. In the majority of instances the pulse wave strikes the finger forcibly with a quick jerking impulse, and immediately recedes or collapses. The characters of this are sometimes best appreciated by grasping the arm above the wrist and holding it up. Moreover, the pulse of aortic regurgitation is usually retarded or delayed—i. e., there is an appreciable interval between the beat of the heart and the pulsation in the radial artery, which varies according to the extent of the incompetence. On auscultation a double murmur may be heard in the carotids and subclavians when it is present at the aortic orifice. Occasionally in the carotid the second sound is distinctly audible when absent at the aortic cartilage. Indeed, according to Broadbent, it is at the carotid that we must listen for the second aortic sound, for when heard it indicates that the regurgitation is small in amount, and is consequently a very favorable prognostic element. In the femoral artery a double murmur also may be heard sometimes, as pointed out by Duroziez.

Aortic insufficiency may for years be fully compensated. Persons do not necessarily suffer any inconvenience, and the condition is often found accidentally. So long as the hypertrophy just equalizes the valvular defect there may be no symptoms and the individual may even take moderately heavy exercise without experiencing sensations of distress about the

heart. The cases which last the longest are those in which the insufficiency follows endocarditis and is not a part of a general arterio-sclerosis. The age of the patient too, at the time the lesion is acquired, is a most important consideration, as in youth the heart is much more prone to take on compensatory changes. Coexistent lesions of the mitral valves tend early to disturb the compensation. It has scarcely been sufficiently recognized by the profession at large that pure aortic insufficiency is consistent with years of average health and with a tolerably active life. I know several physicians with aortic insufficiency who have been able to carry on for years large and somewhat onerous practices. One of them since the establishment of insufficiency has passed successfully through two attacks of acute rheumatism. In a large hospital practice, scarcely a month passes without the discovery of a case of aortic insufficiency in connection with some other affection.

With the onset of myocardial changes, with increasing degeneration of the arteries, particularly with a progressive sclerosis of the arch and involvement of the orifices of the coronary arteries, the compensation becomes disturbed. In advanced cases the changes about the aortic ring may be associated with alterations in the cardiac nerves and ganglia, and so introduce an important factor.

3. Aortic Stenosis.

Narrowing or stricture of the aortic orifice is not nearly so common as insufficiency. The two conditions, as already stated, may occur together, however, and probably in almost every case of stenosis there is some leakage.

Etiology and Morbid Anatomy.—In the milder grades there is adhesion between the segments, which are so stiffened that during systole they cannot be pressed back against the aortic wall. The process of cohesion between the segments may go on without great thickening, and produce a condition in which the orifice is guarded by a comparatively thin membrane, on the aortic face of which may be seen the primitive raphes separating the sinuses of Valsalva. In some instances this membrane is so thin and presents so few traces of atheromatous or sclerotic changes that the condition looks as if it had originated during fœtal life. More commonly the valve segments are thickened and rigid, and have a cartilaginous hardness. In advanced cases they may be represented by stiff, calcified masses obstructing the orifice, through which a circular or slit-like passage can be seen. The older the patient the more likely it is that the valves will be rigid and calcified.

We may speak of a relative stenosis of the aortic orifice when with normal valves and ring the aorta immediately beyond is greatly dilated. A stenosis due to involvement of the aortic ring in sclerotic and calcareous changes without lesion of the valves is referred to by some authors. I have never met with an instance of this kind. A subvalvular stenosis, the result of endocarditis in the mitro-sigmoidean sinus, usually occurs as the result of fœtal endocarditis. In comparison with aortic insufficiency, stenosis is a rare disease. It is usually met with at a more advanced period of life than

insufficiency, and the most typical cases of it are found associated with extensive calcareous changes in the arterial system in old men.

When gradually produced and when there is not much insufficiency the dilatation of the left ventricle may be slight, though I think that in all cases it does occur. The walls of the ventricles become hypertrophied, and we see in this condition the most typical instances of what is called concentric hypertrophy, in which, without much, if any, enlargement of the cavity, the walls are greatly thickened, in contradistinction to the so-called eccentric hypertrophy, in which, with the increase in the thickness of the walls, the chamber itself is greatly dilated. There may be no changes in the other cardiac cavities if compensation is well maintained; but with its failure come dilatation, impeded auricular discharge, pulmonary congestion, and increased work for the right heart. The arterial changes are, as a rule, not so marked as in aortic insufficiency, for the walls have not to withstand the impulse of a greatly increased blood-wave with each systole. On the contrary, the amount of blood propelled through the narrow orifice may be smaller than normal, though when compensation is fully established the pulse-wave may be of medium volume.

Symptoms.—Physical Signs.—*Inspection* may fail to reveal any area of cardiac impulse. Particularly is this the case in old men with rigid chest walls and large emphysematous lungs. Under these circumstances there may be a high grade of hypertrophy without any visible impulse. Even when the apex beat is visible, it may be, as Traube pointed out, feeble and indefinite. In many cases the apex is seen displaced downward and outward, and the impulse looks strong and forcible.

Palpation reveals in many cases a thrill at the base of the heart of maximum force in the aortic region. With no other condition do we meet with thrills of greater intensity. The apex beat may not be palpable under the conditions above mentioned, or there may be a slow, heaving, forcible impulse.

Percussion never gives the same wide area of dulness as in aortic insufficiency. The extent of it depends largely on the state of the lungs, whether emphysematous or not.

Auscultation.—A systolic murmur of maximum intensity at the aortic cartilage, and propagated into the great vessels, is present in aortic stenosis, but is by no means pathognomonic. One of the last lessons learned by the student of physical diagnosis is to recognize the fact that this systolic murmur is only in comparatively rare cases produced by decided narrowing of the aortic orifice. Roughening of the valves, or the intima of the aorta, and hæmic states are much more frequent causes. In aortic stenosis the murmur often has a much harsher quality, is louder, and is more frequently musical than in the conditions just mentioned. When compensation fails and the ventricle is dilated and feeble, the murmur may be soft and distant. The second sound is rarely heard at the aortic cartilage, owing to the thickening and stiffness of the valve. A diastolic murmur is not uncommon, but in many cases it cannot be heard. Occasionally, as noted by W. H. Dickinson, there is a musical murmur of greatest intensity in the region of the apex, due probably to a slight regurgitation at high pressure through

the mitral valves. The pulse in pure aortic stenosis is small, usually of good tension, well sustained, regular, and perhaps slower than normal.

The condition may be latent for an indefinite period, as long as the hypertrophy is maintained. Early symptoms are those due to defective blood-supply to the brain, dizziness, and fainting. Palpitation, pain about the heart, and anginal symptoms are not so marked as in insufficiency. With degeneration of the heart-muscle and dilatation relative insufficiency of the mitral valve is established, and the patient may present all the features of engorgement in the lesser and systemic circulations, with dyspnœa, cough, rusty expectoration, and the signs of anasarca in the lower part of the body. Many of the cases in old people, without presenting any dropsy, have symptoms pointing rather to general arterial disease. Cheyne-Stokes breathing is not uncommon with or without signs of uræmia.

Diagnosis.—With an extremely rough or musical murmur of maximum intensity at the aortic region and signs of hypertrophy of the left ventricle, a thrill, and especially a hard, slow pulse of moderate volume and fairly good tension, which in sphygmographic tracing gives a curve of slow rise, a broad well-sustained summit and slow decline, a diagnosis of aortic stenosis can be made with some degree of probability, particularly if the subject is an old man. Mistakes are common, however, and a roughened or calcified valve segment, or, in some instances, a very roughened and prominent calcified plate in the aorta, and hypertrophy associated with renal disease, may produce similar symptoms.

Let me repeat that a murmur of maximum intensity at the aortic cartilage is of no importance in itself as a diagnostic sign of stenosis. Roughening of the valve, sclerosis of the intima of the arch, and anæmia are conditions more frequently associated with a systolic murmur in this region. Seldom is there difficulty in distinguishing the murmur due to anæmia, since it is rarely so intense and is not associated with thrill or with marked hypertrophy of the left ventricle. In aortic insufficiency a systolic murmur is usually present, but has neither the intensity nor the musical quality, nor is it accompanied with a thrill. With roughening and dilatation of the ascending aorta the murmur may be very harsh or musical; but the existence of a second sound, accentuated and ringing in quality, is usually sufficient to differentiate this condition.

4. Mitral Incompetency.

Etiology.—Insufficiency of the mitral valve results from: (*a*) Changes in the segments whereby they are contracted and shortened, usually combined with changes in the chordæ tendineæ, or with more or less narrowing of the orifice. (*b*) As a result of changes in the muscular walls of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or changes in the muscular substance, so that the segments are imperfectly coapted during the systole—muscular incompetency. The common lesions producing insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordæ tendineæ, and union of the edges of the segments, so that in a

majority of the instances there is not only insufficiency, but some grade of narrowing as well. Except in children, we rarely see the mitral leaflets curled and puckered without narrowing of the orifice. Calcareous plates at the base of the valve may prevent perfect closure of one of the segments. In long-standing cases the entire mitral structures are converted into a firm calcareous ring. From this valvular insufficiency the other condition of muscular incompetency must be carefully distinguished. It is met with in all conditions of extreme dilatation of the left ventricle, and also in weakening of the muscles in prolonged fevers and in anæmia.

Morbid Anatomy.—The effects of incompetency of the mitral segment upon the heart and circulation are as follows: (*a*) The imperfect closure allows a certain amount of blood to regurgitate from the ventricle into the auricle, so that at the end of auricular diastole this chamber contains not only the blood which it has received from the lungs, but also that which has regurgitated from the left ventricle. This necessitates dilatation, and, as increased work is thrown upon it in expelling the augmented contents, hypertrophy as well.

(*b*) With each systole of the left auricle a larger volume of blood is forced into the left ventricle, which also dilates and subsequently becomes hypertrophied.

(*c*) During the diastole of the left auricle, as blood is regurgitated into it from the left ventricle, the pulmonary veins are less readily emptied. In consequence the right ventricle expels its contents less freely, and in turn becomes dilated and hypertrophied.

(*d*) Finally, the right auricle also is involved, its chamber is enlarged, and its walls are increased in thickness.

(*e*) The effect upon the pulmonary vessels is to produce dilatation both of the arteries and veins—often in long-standing cases, atheromatous changes; the capillaries are distended, and ultimately the condition of brown induration is produced. Perfect compensation may be effected, chiefly through the hypertrophy of both ventricles, and the effect upon the peripheral circulation may not be manifested for years, as a normal volume of blood is discharged from the left heart at each systole. The time comes, however, when, owing either to increase in the grade of the incompetency or to failure of the compensation, the left ventricle is unable to send out its normal volume into the aorta. Then there is overfilling of the left auricle, engorgement in the lesser circulation, embarrassed action of the right heart, and congestion in the systemic veins. For years this somewhat congested condition may be limited to the lesser circulation, but finally the right auricle becomes dilated, the tricuspid valves incompetent, and the systemic veins are engorged. This gradually leads to the condition of cyanotic induration in the viscera and, when extreme, to dropsical effusion.

Muscular incompetency, due to impaired nutrition of the mitral and papillary muscles, is rarely followed by such perfect compensation. There may be in acute destruction of the aortic segments an acute dilatation of the left ventricle with relative incompetency of the mitral segments, great dilatation of the left auricle, and intense engorgement of the lungs, under

which circumstances profuse hæmorrhage may result. In these cases there is little chance for the establishment of compensation. In cases of hypertrophy and dilatation of the heart, without valvular lesions, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme and lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy, which cannot be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic Bright's disease the hypertrophy of the left ventricle may gradually fail, leading, in the later stages, to relative insufficiency of the mitral valve, and the production of a condition of pulmonary and systemic congestion, similar to that induced by the most extreme grade of lesion of the valve itself. Adherent pericardium, especially in children, may lead to like results.

Symptoms.—During the development of the lesion, unless the incompetency comes on acutely in consequence of rupture of the valve segment or of ulceration, the compensatory changes go hand in hand with the defect, and there are no subjective symptoms. So, also, in the stage of perfect compensation, there may be the most extreme grade of mitral insufficiency with enormous hypertrophy of the heart, yet the patient may not be aware of the existence of heart trouble, and may suffer no inconvenience except perhaps a little shortness of breath on exertion or on going upstairs. It is only when from any cause the compensation has not been perfectly effected, or, having been so, is broken abruptly or gradually, that the patients begin to be troubled. The symptoms may be divided into two groups:

(*a*) The minor manifestations while compensation is still good. Patients with extreme incompetency often have a congested appearance of the face, the lips and ears have a bluish tint, and the venules on the cheeks may be enlarged, which in many cases is very suggestive. In long-standing cases, particularly in children, the fingers may be clubbed, and there is shortness of breath on exertion. This is one of the most constant features in mitral insufficiency, and may exist for years, even when the compensation is perfect. Owing to the somewhat congested condition of the lungs these patients have a tendency to attacks of bronchitis or hæmoptysis. There may also be palpitation of the heart. As a rule, however, in well-balanced lesions in adults, this period of full compensation or latent stage is not associated with symptoms which call the attention to an affection of the heart, and with care the patient may reach old age in comparative comfort without being compelled to curtail seriously his pleasures or his work.

(*b*) Sooner or later comes a period of disturbed or broken compensation, in which the most intense symptoms are those of venous engorgement. There are palpitation, weak, irregular action of the heart, and signs of dilatation. Dyspnœa is an especial feature, and there may be cough. A distressing symptom is the cardiac "sleep-start," in which, just as the patient falls asleep, he wakes gasping and feeling as if the heart was stopping. There is usually a slight cyanosis, and even a jaundiced tint to the skin. The most marked symptoms, however, are those of venous stasis. The

overfilling of the pulmonary vessels accounts in part for the dyspnœa. There is cough, often with bloody or watery expectoration, and the alveolar epithelium containing brown pigment-grains is abundant. Dropsical effusion usually sets in, beginning in the feet and extending to the body and the serous sacs. The liver is enlarged, and there are signs of portal congestion, gastric irritation, and catarrh of the stomach and intestines. The urine is usually scanty and albuminous, and contains tube-casts and sometimes blood-corpuscles. With judicious treatment the compensation may be restored and all the serious symptoms may pass away. Patients usually have recurring attacks of this kind, and die of a general dropsy; or there is progressive dilatation of the heart, and death from asystole. Sudden death in these cases is rare.

Physical Signs.—*Inspection.*—In children the præcordia may bulge and there may be a large area of visible pulsation. The apex beat is to the left of the nipple, in some cases in the sixth interspace, in the anterior axillary line. There may be a wavy impulse in the cervical veins which are often full, particularly when the patient is recumbent.

Palpation.—A thrill is rare; when present it is felt at the apex, often in a limited area. The force of the impulse may depend largely upon the stage in which the case is examined. In full compensation it is forcible and heaving; when the compensation is disturbed, usually wavy and feeble.

Percussion.—The dulness is increased, particularly in a lateral direction. There is no disease of the valves which produces, in long-standing cases, a more extensive transverse area of heart dulness. It does not extend so much upward along the left margin of the sternum as beyond the right margin and to the left of the nipple line.

Auscultation.—At the apex there is a systolic murmur which wholly or partly obliterates the first sound. It is loudest here, and has a blowing, sometimes musical character, particularly toward the latter part. The murmur is transmitted to the axilla and may be heard at the back, in some instances over the entire chest. There are cases in which, as pointed out by Naunyn, the murmur is heard best along the left border of the sternum. Usually in diastole at the apex the loudly transmitted second sound may be heard. Occasionally there is also a soft, sometimes a rough or rumbling presystolic murmur. As a rule, in cases of extreme mitral insufficiency from valvular lesion with great hypertrophy of both ventricles, there is heard only a loud blowing murmur during systole. A murmur of mitral insufficiency may vary a great deal according to the position of the patient. It may be present in the recumbent and absent in the erect posture. In cases of dilatation, particularly when dropsy is present, there may be heard at the ensiform cartilage and in the lower sternal region a soft systolic murmur due to tricuspid regurgitation. An important sign on auscultation is the accentuated pulmonary second sound. This is heard to the left of the sternum in the second interspace, or over the third left costal cartilage.

The pulse in mitral insufficiency, during the period of full compensation, may be full and regular, often of low tension. Usually with the first onset of the symptoms the pulse becomes irregular, a feature which then

dominates the case throughout. There may be no two beats of equal force or volume. Often after the disappearance of the symptoms of failure of compensation the irregularity of the pulse persists.

The three important physical signs then of mitral regurgitation are: (*a*) Systolic murmur of maximum intensity at the apex, which is propagated to the axilla and heard at the angle of the scapula; (*b*) accentuation of the pulmonary second sound; (*c*) evidence of enlargement of the heart, particularly the increase in the transverse diameter, due to hypertrophy of both right and left ventricles.

Diagnosis.—There is rarely any difficulty in the diagnosis of mitral insufficiency. The physical signs just referred to are quite characteristic and distinctive. Two points are to be borne in mind. First, a murmur, systolic in character, and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate incompetency of the mitral valve. There is heard in this region a large group of what are termed accidental murmurs, the precise nature of which is still doubtful. They are probably formed, however, in the ventricle, and are not associated with hypertrophy, or accentuation of pulmonary second sound.

Second, it is not always possible to say whether the insufficiency is due to lesion of the valve segment or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary second sound, nor the hypertrophy assists in the differentiation. The history is sometimes of greater value in this matter than the physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart (in which the systolic murmur may be of the greatest intensity), and the instances of arterio-sclerosis with dilated heart. Balfour and others, however, maintain that organic disease of the mitral leaflets sufficient to produce incompetency is always accompanied with a certain degree of narrowing of the orifice, so that the only unequivocal proof of the actual disease of the mitral valve is the presence of a presystolic murmur.

5. Mitral Stenosis.

Etiology.—Narrowing of the mitral orifice is usually the result of valvular endocarditis occurring in the earlier years of life; very rarely it is congenital. It is very much more common in women than in men—in 63 of 80 cases noted by Duckworth, while in 4,791 autopsies at Guy's Hospital during ten years there were 196 cases, of which 107 were females and 89 males (Samways). This is not easy to explain, but there are at least two factors to be considered. Rheumatism prevails more in girls than in boys and, as is well known, endocarditis of the mitral valve is more common in rheumatism. Chorea, also, as suggested by Barlow, has an important influence, occurring more frequently in girls and being often associated with endocarditis. Of 140 cases of chorea which I examined at a period more than two years subsequent to the attack, 72 had signs of organic heart-disease, among which were 24 instances with the physical signs of mitral stenosis. Anæmia and chlorosis, which are prevalent in girls, have

been regarded as possible factors. In a surprising number of cases no recognizable etiological factor can be discovered. This has been regarded by some writers as favoring the view that many cases are of congenital origin; but it is not improbable that with any of the febrile affections of childhood endocarditis may be associated. Whooping-cough, too, with its terrible strain on the heart-valves, may be accountable for certain cases. Congenital affections of the mitral valve are notoriously rare. While met with at all ages, stenosis is certainly more frequent in young persons.

Morbid Anatomy.—In a majority of instances with the stenosis there is some incompetency; indeed, Balfour maintains that we never find mitral stenosis without some degree of regurgitation. The narrowing results from thickening and contraction of the tissues of the ring, of the valve segments, and of the chordæ tendineæ. The condition varies a good deal according to the amount of atheromatous change. In many cases the curtains are so welded together and the whole valvular region so thickened that the orifice is reduced to a mere chink—Corrigan's button-hole contraction. In other cases the curtains are not much thickened, but narrowing has resulted from gradual adhesion at the edges, and thickening of the chordæ tendineæ, so that from the auricle it looks cone-like—the so-called funnel-shaped variety of stenosis. The instances in which the valve segments are very slightly deformed, but in which the orifice is considerably narrowed, are regarded by some as possibly of congenital origin. Occasionally the curtains are in great part free from disease, but the narrowing results from large calcareous masses, which project into them from the ring. The involvement of the chordæ tendineæ is usually extreme, and the papillary muscles may be inserted directly upon the valve. In moderate grades of constriction the orifice will admit the tip of the index-finger; in more extreme forms, the tip of the little finger; and occasionally one meets with a specimen in which the orifice seems almost obliterated, as in a case which came under my notice, which only admitted a medium-sized Bowman's probe.

The heart in mitral stenosis is not greatly enlarged, rarely weighing more than 14 or 15 ounces. Occasionally, in an elderly person, it may seem only slightly, if at all, enlarged, and again there are instances in which the weight may reach as much as 20 ounces. The left ventricle is usually small, and may look very small in comparison with the right ventricle, which forms the greater portion of the apex. In cases in which with the narrowing there is very considerable incompetency the left ventricle may be moderately dilated and hypertrophied.

These changes gradually induced are associated with secondary alterations of great importance in the heart. The left auricle discharges its blood with greater difficulty and in consequence dilates, and its walls reach three or four times their normal thickness. Although the auricle is by structure unfitted to compensate an extreme lesion, the probability is that for some time during the gradual production of stenosis, the increasing muscular power of the walls is sufficient to counterbalance the defect. Samways found in 36 cases of well-marked stenosis the auricle hypertrophied in 26, dilatation coexisting in 14. Eventually the tension is increased in the pul-

monary circulation, owing to impeded outflow from the veins. To overcome this the right ventricle undergoes dilatation and hypertrophy, and upon this chamber falls the work of equalizing the circulation. Relative incompetency of the tricuspid and congestion of the systemic veins at last supervene.

It is not uncommon at the examination to find white thrombi in the appendix of the left auricle. Occasionally a large part of the auricle is occupied by an ante-mortem thrombus. Still more rarely the remarkable ball thrombus is found, in which a globular concretion, varying in size from a walnut to a small egg, lies free in the auricle, two examples of which have come under my observation.

Symptoms.—Physical Signs.—*Inspection.*—In children the lower sternum and the fifth and sixth left costal cartilages are often prominent, owing to hypertrophy of the right ventricle. The apex beat may be ill-defined. Usually, it is not dislocated far beyond the nipple line, and the chief impulse is over the lower sternum and adjacent costal cartilages. Often in thin-chested persons there is pulsation in the third and fourth left interspaces close to the sternum. When compensation fails, the præcordial impulse is much feebler, and in the veins of the neck there may be marked systolic regurgitation.

Palpation reveals in a majority of the cases a characteristic, well-defined fremitus or thrill, which is best felt, as a rule, in the fourth or fifth interspace within the nipple line. It is of a rough, grating quality, often peculiarly limited in area, most marked during expiration, and can be felt to terminate in a sharp, sudden shock, synchronous with the impulse. This most characteristic of physical signs is pathognomonic of narrowing of the mitral orifice, and is perhaps the only instance in which the diagnosis of a valvular lesion can be made by palpation alone. The cardiac impulse is felt most forcibly in the lower sternum and in the fourth and fifth left interspaces. The impulse is felt very high in the third and fourth interspaces, or in rare cases even in the second, and it has been thought that in the latter interspace the impulse is due to pulsation of the auricle. It is always the impulse of the conus arteriosus of the right ventricle; even in the most extreme grades of mitral stenosis, there is never such tilting forward of the auricle or its appendix as would enable it to produce an impression on the chest wall.

Percussion gives an increase in the cardiac dulness to the right of the sternum and along the left margin; not usually a great increase beyond the nipple line, except in extreme cases, when the transverse dulness may reach from 5 cm. beyond the right margin of the sternum to 10 cm. beyond the nipple line.

Auscultation.—In the mitral area, usually to the inner side of the apex beat and often in a very limited region, is heard a rough, vibratory or purring murmur, which terminates abruptly in the first sound. By combining palpation and auscultation the purring murmur is found to be synchronous with the thrill and the loud shock with the first sound. This is the presystolic murmur, about the time and mode of production of which so much discussion has occurred. I hold with those who regard it as occur-

ring during the auricular systole. In whatever way produced, it remains one of the most distinctive and characteristic of murmurs and its presence is positively indicative of narrowing of the mitral orifice. The sole exception to this statement is the Flint murmur already referred to in aortic incompetency. Once, in a case of enormous enlargement of the spleen, with dropsy, in which the heart was greatly pushed up, I heard a presystolic murmur of rough quality, and the mitral valves were found post mortem to be normal. The presystolic murmur may occupy the entire period of the diastole, or the middle or only the latter half, corresponding to the auricular systole. The difference may sometimes be noted between the first and second portions of the murmur, when it occupies the entire time. Often there is a peculiar rumbling or echoing quality, which in some instances is very limited and may be heard only over a single bell-space of the stethoscope. A systolic murmur may be heard at the apex or along the left sternal border, often of extreme softness and audible only when the breath is held. Sometimes the systolic murmur is loud and distinct and is transmitted to the axilla. The second sound in the second left interspace is loudly accentuated, sometimes reduplicated. It may be transmitted far to the left and be heard with great clearness beyond the apex. In uncomplicated cases of mitral stenosis there are usually no murmurs audible at the aortic region, at which spot the second sound is less intense than at the pulmonary area. In the lower sternum and to the right a tricuspid murmur is sometimes heard in advanced cases. Other points to be noted are the following: The unusually sharp, clear first sound which follows the presystolic murmur, the cause of which is by no means easy to explain. It can scarcely be a valvular sound produced chiefly at the mitral orifice, since it may be heard with great intensity in cases in which the valves are rigid and calcified. It has been suggested by W. S. Fenwick and Overend that it is a loud "snap" of the tricuspid valves caused by the powerful contraction of the greatly hypertrophied right ventricle. Broadbent's explanation is as follows: "Owing to the narrowing of the mitral orifice there is not time in the diastolic interval for a sufficient amount of blood to flow into the left ventricle to completely fill it. At the commencement of systole, therefore, the ventricular cavity is not fully distended with blood, so that the muscular walls at the first moment of their contraction meet with no resistance; then closing down rapidly, they are suddenly brought up and made tense as they encounter the contained blood. This sudden tension and abbreviated systole may thus account for the short first sound." The valvular sound may be audible at a distance, as one sits at the bedside of the patient (Graves).

These physical signs, it is to be borne in mind, are characteristic only of the stage in which compensation is maintained. Finally there comes a period in which, with rupture of compensation, the presystolic murmur disappears and there is heard in the apex region a sharp first sound, or sometimes a gallop rhythm. The marked systolic shock may be present after the disappearance of the thrill and the characteristic murmur. Under treatment, with gradual recovery of compensation, probably with increasing vigor of contraction of the right ventricle and left auricle, the pre-

systolic murmur reappears. In cases seen at this stage of the disease the nature of the valve lesion may be entirely overlooked.

Stenosis of the mitral valve may for years be efficiently compensated by the hypertrophy of the right ventricle. Many persons with the characteristic physical signs of this lesion present no symptoms. They may for years perhaps be short of breath on going upstairs, but are able to pass through the ordinary duties of life without discomfort. The pulse is smaller in volume than normal, but may be perfectly regular. A special danger of this stage is the recurring endocarditis. Vegetations may be whipped off into the circulation and, blocking a cerebral vessel, may cause hemiplegia or aphasia, or both. This, unfortunately, is not an uncommon sequence in women. Patients with mitral stenosis may survive this accident for an indefinite period. A woman, above seventy years of age, died in one of my wards at the Philadelphia Hospital, who had been in the almshouse, hemiplegic, for more than thirty years. The heart presented an extreme grade of mitral stenosis which had probably existed at the time of the hemiplegic attack.

Pressure of the enlarged auricle on the left recurrent laryngeal nerve, causing paralysis of the vocal cord on the corresponding side, has been described by Ortner and by Herrick. I have met with two instances. It is a point to be borne in mind, as the diagnosis of aneurism of the arch of the aorta may be made.

Failure of compensation brings in its train the group of symptoms which have been discussed under mitral insufficiency. Briefly enumerated they are: Rapid and irregular action of the heart, shortness of breath, cough, signs of pulmonary engorgement, and very frequently hæmoptysis. Attacks of this kind may recur for years. Bronchitis or a febrile attack may cause shortness of breath or slight blueness. Inflammatory affections of the lungs or pleura seriously disturb the right heart, and these patients stand pneumonia very badly. Many, perhaps a majority of cases of mitral stenosis, do not have dropsy. The liver may be greatly enlarged, and in the late stages ascites is not uncommon, particularly in children. General anasarca is most frequently met with in those cases in which there is secondary narrowing of the tricuspid orifice (Broadbent).

6. Tricuspid Valve Disease.

(*a*) **Tricuspid Regurgitation.**—Occasionally this results from acute or chronic endocarditis with puckering; more commonly the condition is one of relative insufficiency, and is secondary to lesions of the valves on the left side, particularly of the mitral. It is met with also in all conditions of the lungs which cause obstruction to the circulation, such as cirrhosis and emphysema, particularly in combination with chronic bronchitis. The symptoms are those of obstruction in the lesser circulation with venous congestion in the systemic veins, such as has already been described in connection with mitral insufficiency. The signs of this condition are:

(1) Systolic regurgitation of the blood into the right auricle and the transmission of the pulse-wave into the veins of the neck. If the regurgi-

tation is slight or the contraction of the ventricle is feeble there may be no venous throbbing, but in other cases there is marked systolic pulsation in the cervical veins. That in the right jugular is more forcible than that in the left. It may be seen both in the internal and the external vein, particularly in the latter. Marked pulsation in these veins occurs only when the valves guarding them become incompetent. Slight oscillations are by no means uncommon, even when the valves are intact. The distention is sometimes enormous, particularly in the act of coughing, when the right jugular at the root of the neck may stand out, forming an extraordinary prominent ovoid mass. Occasionally the regurgitant pulse-wave may be widely transmitted and be seen in the subclavian and axillary veins, and even in the subcutaneous veins over the shoulder, or, as in a case recently under observation, in the superficial mammary veins.

Regurgitant pulsation through the tricuspid orifice may be transmitted to the inferior cava, and so to the hepatic veins, causing a systolic distention of the liver. This is best appreciated by bimanual palpation, placing one hand over the fifth and sixth costal cartilages and the other in the lateral region of the liver in the mid-axillary line. The rhythmical expansile pulsation may be readily distinguished, as a rule, from the systolic depression of the liver due to communicated pulsation from the left ventricle.

(2) The second important sign of tricuspid regurgitation is the occurrence of a systolic murmur of maximum intensity in the lower sternum. It is usually a soft, low murmur, often to be distinguished from a coexisting mitral murmur by differences in quality and pitch, and may be heard to the right as far as the axilla. Sometimes it is very limited in its distribution.

Together these two signs positively indicate tricuspid regurgitation. In addition, the percussion usually shows increase in the area of dulness to the right of the sternum, and the impulse in the lower sternal region is forcible. In the great majority of cases the symptoms are those of the associated lesions. In cirrhosis of the lung and in chronic emphysema the failure of compensation of the right ventricle with insufficiency of the tricuspid not infrequently leads either to acute asystole or to gradual failure with cardiac dropsy.

(*b*) **Tricuspid Stenosis.**—This interesting condition may be either congenital or acquired. The congenital cases are not uncommon, and are associated usually with other valvular defects which cause early death. The acquired form is not very infrequent. Bedford Fenwick collected 46 observations, of which 41 were in women. Leudet * has analyzed 117 cases. Of 101 of these in which the ages were mentioned, 80 were in women and 21 in men. A great majority of the cases were in adults, only 8 being between the ages of ten and twenty. Its rarity as an isolated condition may be gathered from the fact that of 114 autopsies, in 11 only was the lesion confined to this valve. In 21 the tricuspid, mitral, and aortic seg-

* Paris Thesis, 1888.

ments were involved, and in 78 the tricuspid and mitral. Practically the condition is almost always secondary to lesions of the left heart.

The physical signs are sometimes characteristic. For instance, a presystolic thrill has been noted by several observers. The percussion shows dulness to be increased, particularly to the right of the sternum. On auscultation a presystolic murmur has been determined in certain cases, and is heard best at the root of the ensiform cartilage, or a little to the right of it. Of general symptoms, cyanosis of the face and lips is very common, and in the late stages, when dropsy supervenes, it is apt to be intense. The lesion is interesting chiefly because it forms one of the most serious complications of mitral stenosis.

7. Pulmonary Valve Disease.

Murmurs in the region of the pulmonary valves are extremely common; lesions of the valves are exceedingly rare. Balfour has well called the pulmonic area the region of romance. A systolic murmur is heard here under many conditions—(1) very often in health, in thin-chested persons, particularly in children, during expiration and in the recumbent posture; (2) when the heart is acting rapidly, as in fever and after exertion; (3) it is a favorite situation of the cardio-respiratory murmur; (4) in anæmic states; and (5) as mentioned previously, the systolic murmur of mitral insufficiency may be transmitted along the left sternal margin. Actual lesions of the valves of the pulmonary artery are rare.

(*a*) *Stenosis* is almost invariably a congenital anomaly. It constitutes one of the most important of the congenital cardiac affections. The valve segments are usually united, leaving a small, narrow orifice. In the adult cases occasionally occur. In Case 608 of my post-mortem records there was extreme stenosis in a girl of eighteen, owing to great thickening and adhesion of the segments, and there were also numerous vegetations. The orifice was only 2 mm. in diameter. The congenital lesion is commonly associated with patency of the ductus Botalii and imperfection of the ventricular septum. There may also be tricuspid stenosis.

The physical signs are extremely uncertain. There may be a systolic murmur with a thrill heard best to the left of the sternum in the second intercostal space. This murmur may be very like a murmur of aortic stenosis, but is not transmitted into the vessels. Naturally the pulmonary second sound is weak or obliterated, or may be replaced by a diastolic murmur. Usually there is hypertrophy of the right heart.

(*b*) *Pulmonary Insufficiency.*—This rare affection is occasionally due to congenital malformation, particularly fusion of two of the segments. It is sometimes present, as Bramwell has shown, in cases of malignant endocarditis. Barie has collected 58 cases.

The physical signs are those of regurgitation into the right ventricle, but, as a rule, it is difficult to differentiate the murmur from that of aortic insufficiency, though the maximum intensity may be in the pulmonary area. The absence of the vascular features of aortic insufficiency is suggestive. Both Gibson and Graham Steell have called attention to the pos-

sibility of leakage through these valves in cases of great increase of pressure in the pulmonary artery, and to a soft diastolic murmur heard under these circumstances, which Steell calls "the murmur of high pressure in the pulmonary artery."

8. Combined Valvular Lesions.

These are extremely common. The mitral and aortic segments may be affected together; next in frequency comes the combination of mitral and tricuspid lesions; and then of aortic, mitral, and tricuspid. Aortic insufficiency or aortic stenosis is more frequently combined with mitral incompetency than aortic stenosis with mitral stenosis, or mitral stenosis with aortic insufficiency. In children the most common combination is aortic and mitral insufficiency. In adults, mitral insufficiency with thickening of the aortic valves and slight narrowing is perhaps the most common.

The diagnosis rests upon the character of the murmurs and the state of the chambers as regards hypertrophy and dilatation.

Prognosis in Valvular Disease.—The question is entirely one of efficient compensation. So long as this is maintained the patient may suffer no inconvenience, and even with the most serious forms of valve lesion the function of the heart may be little, if at all, disturbed.

Practitioners who are not adepts in auscultation and feel unable to estimate the value of the various heart murmurs should remember that the best judgment of the conditions may be gathered from inspection and palpation. With an apex beat in the normal situation and regular in rhythm the auscultatory phenomena may be practically disregarded.

As Sir Andrew Clark states, a murmur *per se* is of little or no moment in determining the prognosis in any given case. There is a large group of patients who present no other symptoms than a systolic murmur heard over the body of the heart, or over the apex, in whom the left ventricle is not hypertrophied, the heart rhythm is normal, and who may not have had rheumatism. Indeed, the condition is accidentally discovered, often during examination for life insurance. I know cases of this kind which have persisted unchanged for more than fifteen years. Among the conditions influencing prognosis are:

(*a*) *Age.*—Children under ten are bad subjects. Compensation is well effected, and they are free from many of the influences which disturb compensation in adults. The coronary arteries are healthy, and nutrition of the heart-muscle can be readily maintained. Yet, in spite of this, the outlook in cardiac lesions developing in very young children is usually bad. One reason is that the valve lesion itself is apt to be rapidly progressive, and the limit of cardiac reserve force is in such cases early reached. There seems to be proportionately a greater degree of hypertrophy and dilatation. Among other causes of the risks of this period are to be mentioned insufficient food in the poorer classes, the recurrence of rheumatic attacks, and the existence of pericardial adhesions. The outlook in a child who can be carefully supervised and prevented from damaging himself by overexertion is naturally better than in one who is constantly overtasking his muscles.

The valvular lesions which develop at, or subsequent to, the period of puberty are more likely to be permanently and efficiently compensated. Sudden death from heart-disease is very rare in children.

(*b*) *Sex.*—Women bear valve lesions, as a rule, better than men, owing partly to the fact that they live quieter lives, partly to the less common involvement of the coronary arteries, and to the greater frequency of mitral lesions. Pregnancy and parturition are disturbing factors, but are, I think, less serious than some writers would have us believe.

(*c*) *Valve affected.*—The relative prognosis of the different valve lesions is very difficult to estimate. Each case must, therefore, be judged on its own merits. Aortic insufficiency is unquestionably the most serious; yet for years it may be perfectly compensated. Favorable circumstances in any case are the moderate grade of hypertrophy and dilatation, the absence of all symptoms of cardiac distress, and the absence of extensive arterio-sclerosis and of angina. The prognosis rests in reality with the condition of the coronary arteries. Rheumatic lesions of the valves, inducing insufficiency, are less apt to be associated with endarteritis at the root of the aorta; and in such cases the coronary arteries may escape for years. I know a physician, now about forty-three years of age, who, when sixteen, had his first attack of rheumatism, which involved the aortic segments. He has had two subsequent attacks of rheumatism, but with care has been able to live a comfortable and fairly active life. On the other hand, when the aortic insufficiency is only a part of an extensive arterio-sclerosis at the root of the aorta, the coronary arteries are almost invariably involved, and the outlook in such cases is much more serious. Sudden death is not uncommon, either from acute dilatation during some exertion, or, more frequently, from blocking of one of the branches of the coronary arteries. The liability of this form to be associated with angina pectoris also adds to its severity. Aortic stenosis is a comparatively rare lesion, most commonly met with in middle-aged or elderly men, and is, as a rule, well compensated. In Broadbent's series of cases, in which autopsy showed definite aortic narrowing, forty years was the average age at death, and the oldest was but fifty-three.

In mitral lesions the outlook on the whole is much more favorable than in aortic insufficiency. Mitral insufficiency, when well compensated, carries with it a better prognosis than mitral stenosis. Practically it is the only valvular disease we meet with in patients over threescore years. It must be borne in mind that the cases which last the longest are those in which the valve orifice is more or less narrowed, as well as incompetent. There is, in reality, no valve lesion so poorly compensated and so rapidly fatal as that in which the mitral segments are gradually curled and puckered until they form a narrow strip around a wide mitral ring—a condition specially seen in children. There are many cases of mitral insufficiency in which the defect is thoroughly balanced for thirty or even forty years, without distress or inconvenience. Even with great hypertrophy and the apex beat almost in the mid-axillary line, there may be little or no distress, and the compensation may be most effective. Women may pass safely through repeated pregnancies, though here they are liable to accidents asso-

ciated with the severe strain. I have had under my care for many years a patient who had her first attack of rheumatism at the age of fifteen, when she already had a well-marked mitral murmur. She first came under my observation, twenty-four years ago, with signs of hypertrophy of the left ventricle and a loud systolic murmur. She has had no cardiac disturbance whatever, though she has lived a very active life, has been unusually vigorous, has borne eleven children, and has passed through three subsequent attacks of rheumatism.

In mitral stenosis the prognosis is usually regarded as less favorable. My own experience has led me, however, to place this lesion almost on a level, particularly in women, with the mitral insufficiency. It is found very often in persons in perfect health, who have had neither palpitation nor signs of heart-failure, and who have lived laborious lives. The figures given, too, by Broadbent indicate that the date of death in mitral stenosis is comparatively advanced. Of 53 cases abstracted from the post-mortem records of St. Mary's Hospital, thirty-three was the age for males, and thirty-seven or thirty-eight for females. These women, too, pass through repeated pregnancies with safety. There are of course those too common accidents, the result of cerebral embolism, which are more likely to occur in this than in other forms.

Hard and fast lines cannot be drawn in the question of prognosis in valvular disease. Every case must be judged separately, and all the circumstances carefully balanced. There is no question which requires greater experience and more mature judgment, and even the most experienced are sometimes at fault.

The following brief summary of the conditions which justify a favorable prognosis embodies the large and varied clinical experience of Sir Andrew Clark: Good general health; just habits of living; no exceptional liability to rheumatic or catarrhal affections; origin of the valvular lesion independently of degeneration; existence of the valvular lesion without change for over three years; sound ventricles, of moderate frequency and general regularity of action; sound arteries, with a normal amount of blood and tension in the smaller vessels; free course of blood through the cervical veins; and, lastly, freedom from pulmonary, hepatic, and renal congestion.

Treatment of Valvular Lesions.—For this purpose the valvular lesion may be divided into the period of progressive development, with establishment and maintenance of hypertrophy, and the period of disturbed compensation.

(*a*) **Stage of Compensation.**—Medicinal treatment at this period is not necessary and is often hurtful. A very common error is to administer cardiac drugs, such as digitalis, on the discovery of a murmur or of hypertrophy. If the lesion has been found accidentally, it may be best not to tell the patient, but rather an intimate friend. Often it is necessary, however, to be perfectly frank in order that the patient may take certain preventive measures. He should lead a quiet, regulated, orderly life, free from excitement and worry, and the risk of sudden death makes it imperative that the patient suffering from aortic disease should be specially warned

against overexertion and hurry. An ordinary wholesome diet in moderate quantities should be taken, tobacco should be interdicted, and stimulants not allowed. Exercise should be regulated entirely by the feelings of the patient. So long as no cardiac distress or palpitation follows, moderate exercise will prove very beneficial. The skin should be kept active by a daily bath. Hot baths should be avoided and the Turkish bath should be interdicted. In the case of full-blooded, somewhat corpulent individuals, an occasional saline purge should be taken. Patients with valvular lesions should not go into very high altitudes. The act of coition has serious risks, particularly in aortic insufficiency. Knowing that the causes which most surely and powerfully disturb the compensation are overexertion, mental worry, and malnutrition, the physician should give suitable instructions in each case. As it is always better to have the co-operation of an intelligent patient, he should, as a rule, be told of the condition, but in this matter the physician must be guided by circumstances, and there are cases in which reticence is the wiser policy.

(*b*) **Stage of Broken Compensation.**—The break may be immediate and final, as when sudden death results from acute dilatation or from blocking of a branch of the coronary artery, or it may be gradual. Among the first indications are shortness of breath on exertion or attacks of nocturnal dyspnœa. These are often associated with impaired nutrition, particularly with anæmia, and a course of iron or change of air may suffice to relieve the symptoms.

Irregularity of the action of the heart cannot always be termed an indication of failing compensation, particularly in instances of mitral disease. It has greater significance in aortic lesions. Serious failure of compensation is indicated by signs of dilatation of the heart, marked cyanosis, the gallop rhythm, or various forms of arrhythmia, with or without the existence of dropsy. Under these circumstances the following measures are to be carried out:

(1) *Rest.*—Disturbed compensation may be completely restored by rest of the body. Both in Montreal and in Philadelphia it was a favorite demonstration in practical therapeutics to show the benign influence of complete rest and quiet on the cardiac dilatation. In many cases with œdema of the ankles, moderate dilatation of the heart, and irregularity of the pulse, the rest in bed, a few doses of the compound tincture of cardamoms, and a saline purge suffice, within a week or ten days, to restore the compensation. One patient, in Ward 11 of the Montreal General Hospital, with aortic insufficiency recovered from four successive attacks of failing compensation with these measures alone.

(2) The relief of the embarrassed circulation.

(*a*) *By Venesection.*—In cases of dilatation, from whatever cause, whether in mitral or aortic lesions or distention of the right ventricle in emphysema, when signs of venous engorgement are marked and when there is orthopnœa with cyanosis, the abstraction of from 20 to 30 ounces of blood is indicated. This is the occasion in which timely venesection may save the patient's life. It is a condition in which I have had most satisfactory results from blood-letting. It is done much better early than late. I have

on several occasions regretted its postponement, particularly in instances of acute dilatation and cyanosis in connection with emphysema.*

(*b*) *By Depletion through the Bowels.*—This is particularly valuable when dropsy is present. Of the various purges the salines are to be preferred, and may be given by Matthew Hay's method. Half an hour to an hour before breakfast from half an ounce to an ounce and a half of Epsom salts may be given in a concentrated form. This usually produces from three to five liquid evacuations. The compound jalap powder in half-drachm doses, or elaterium, may be employed for the same purpose. Even when the pulse is very feeble these hydragogue cathartics are well borne, and they deplete the portal system rapidly and efficiently.

(*c*) *The Use of Remedies which stimulate the Heart's Action.*—Of these, by far the most important is digitalis, which was introduced into practice by Withering. The indication for its use is dilatation; the contra-indication is a perfectly balanced compensatory hypertrophy, such as we see in all forms of valvular disease. Broken compensation, no matter what the valve lesion may be, is the signal for its use. It acts upon the heart, slowing and at the same time increasing the force of the contractions. It acts on the peripheral arteries, raising their tension, so that a steady and equable flow of blood is maintained in the capillaries, which, after all, is the prime aim and object of the circulation. The beneficial effects are best seen in cases of mitral disease with small, irregular pulse and cardiac dropsy. Its effects are not less striking in the dilatation of the left ventricle, in the failing compensation of aortic insufficiency or of arterio-sclerosis. On theoretical grounds it has been urged that its use is not so advantageous in aortic insufficiency, since it prolongs the diastole and leads to greater distention. This need not be considered, and digitalis is just as serviceable in this as in any other condition associated with progressive dilatation; larger doses are often required. It may be given as the tincture or the infusion. In cases of cardiac dropsy, from whatever cause, 15 minims of the tincture or half an ounce of the infusion may be given every three hours for two days, after which the dose may be reduced. Some prefer the tincture, others the infusion; it is a matter of indifference if the drug is good. The urine of a patient taking digitalis should be carefully estimated each day. As a rule, when its action is beneficial, there is within twenty-four hours an increase in the amount; often the flow is very great. Under its use the dyspnœa is relieved, the dropsy gradually disappears, the pulse becomes firmer, fuller in volume, and sometimes, if it has been very intermittent, regular.

Ill effects sometimes follow digitalis. There is no such thing as a cumulative action of the drug manifested by sudden symptoms. Toxic effects are seen in the production of nausea and vomiting. The pulse becomes irregular and small, and there may be two beats of the heart to one of the pulse, which, as pointed out by Broadbent, is found particularly in cases of mitral stenosis when they are under the influence of this drug.

* For illustrative cases from my wards see paper by H. A. Lafleur, Medical News, July, 1891.

The urine is reduced in amount. These symptoms subside on the withdrawal of the digitalis, and are rarely serious. There are patients who take digitalis uninterruptedly for years, and feel palpitation and distress if the drug is omitted. In mitral disease, even when it does good it does not always steady the pulse. There are many cases in which the irregularity is not affected by the digitalis. When the compensation has been re-established the drug may be omitted. When there is dyspnœa on exertion and cardiac distress, from 5 to 10 minims three times a day may be advantageously given for prolonged periods, but the effects should be carefully watched. In cardiac dropsy digitalis should be used at the outset with a free hand. Small doses should not be given, but from the first half-ounce doses of the infusion every three hours, or from 15 to 20 minims of the tincture. There are no substitutes for digitalis.

Of other remedies strophanthus alone is of service. Given in doses of from 5 to 8 minims of the tincture, it acts like digitalis. It certainly will sometimes steady the intermittent heart of mitral valve disease when digitalis fails to do so, but it is not to be compared with this drug when dropsy is present. Convallaria, citrate of caffeine, and *adonis vernalis* and *sparteine* are warmly recommended as substitutes for digitalis, but their inferiority is so manifest that their use is rarely indicated.

There are two valuable adjuncts in the treatment of valvular disease—iron and strychnia. When anæmia is a marked feature iron should be given in full doses. In some instances of failing compensation iron is the only medicine needed to restore the balance. Arsenic is occasionally an excellent substitute, and one or other of them should be administered in all instances of heart-trouble when pallor is present. Strychnia is a heart tonic of very great value. It may be given alone or in combination with the digitalis in 1 or 2 drop doses of the 1-per-cent solution. Alcoholic stimulants in moderation are occasionally useful, especially in tiding over a period of acute cardiac weakness.

Treatment of Special Symptoms. (*a*) *Dropsy.*—The increased arterial tension and activity of the capillary circulation under the influence of digitalis hastens the interstitial lymph flow and favors resorption of the fluid. The hydragogue cathartics, by rapidly depleting the blood, promote, too, the absorption of the fluid from the lymph spaces and the lymph sacs. These two measures usually suffice to rid the patient of the dropsy. In some cases, however, it cannot be relieved, and then Southey's tubes may be used or the legs punctured. If done with care, after a thorough washing of the parts, and if antiseptic precautions are taken, scarification is a very serviceable measure, and should be resorted to more frequently than it is. Canton flannel bandages may be applied on the œdematous legs.

(*b*) *Dyspnœa.*—The patients are usually unable to lie down. A comfortable bed-rest should therefore be provided—if possible, one with lateral projections, so that in sleeping the head can be supported as it falls over. The shortness of breath is associated with dilatation, chronic bronchitis, or hydrothorax. The chest should be carefully examined in all these cases, as hydrothorax of one side or of both is a common cause of shortness of breath. There are cases of mitral regurgitation with recurring hydrothorax

as the sole dropsical symptom, which is relieved, week by week or month by month, by tapping. For the nocturnal dyspnœa, particularly when combined with restlessness, morphia is invaluable and may be given without hesitation. The value of the calming influence of opium in all conditions of cardiac insufficiency is not enough recognized. There are instances of cardiac dyspnœa unassociated with dropsy, particularly in mitral valve disease, in which nitroglycerin is of great service, if given in the 1-per-cent solution in increasing doses. It is especially serviceable in the cases in which the pulse tension is high.

(*c*) *Palpitation and Cardiac Distress.*—In instances of great hypertrophy and in the throbbing which is so distressing in some cases of aortic insufficiency, aconite is of service in doses of from 1 to 3 minims every two or three hours. An ice-bag over the heart or Leiter's coil is also of service in allaying the rapid action and the throbbing. For the pains, which are often so marked in aortic lesions, iodide of potassium in 10-grain doses, three times a day, or the nitroglycerin may be tried. Small blisters are sometimes advantageous. It must be remembered that an important cause of palpitation and cardiac distress is flatulent distention of the stomach or colon, against which suitable measures must be directed.

(*d*) *Gastric Symptoms.*—The cases of cardiac insufficiency which do badly and fail to respond to digitalis are most often those in which nausea and vomiting are prominent features. The liver is often greatly enlarged in these cases; there is more or less stasis in the hepatic vessels, and but little can be expected of drugs until the venous engorgement is relieved. If the vomiting persists, it is best to stop the food and give small bits of ice, small quantities of milk and lime water, and effervescing drinks, such as Apollinaris water and champagne. Creasote, hydrocyanic acid, and the oxalate of cerium are sometimes useful; but, as a rule, the condition is obstinate and always serious.

(*e*) *Cough and Hœmoptysis.*—The former is almost a necessary concomitant of cardiac insufficiency, owing to engorgement of the pulmonary vessels and more or less bronchitis. It is allayed by measures directed rather to the heart than to the lungs. Hæmoptysis in chronic valvular disease is sometimes a salutary symptom. An army surgeon, who was invalided during the late civil war on account of hæmoptysis, supposed to be due to tuberculosis, has since that time had, in association with mitral insufficiency and enlarged heart, many attacks of hæmoptysis. He assures me that his condition is invariably better after the attack. It is rarely fatal, except in some cases of acute dilatation, and seldom calls for special treatment.

(*f*) *Sleeplessness.*—One of the most distressing features of valvular lesions, even in the stage of compensation, is disturbed sleep. Patients may wake suddenly with throbbing of the heart, often in an attack of nightmare. Subsequently, when the compensation has failed, it is also a worrying symptom. The sleep is broken, restless, and frequently disturbed by frightful dreams. Sometimes a dose of the spirits of chloroform or of ether, with half a drachm of spirits of camphor, given in a little hot whisky, will give a quiet night. The compound spirits of ether, Hoffman's anodyne,

though very unpleasant to take, is frequently a great boon in the intermediate period when compensation has partially failed and the patients suffer from restless and sleepless nights. Paraldehyde and amylene hydrate are sometimes serviceable. Urethan, sulphonal, and chloralamide are rarely efficacious, and it is best, after a few trials, particularly if the paraldehyde does not answer, to resort to morphia. It may be given in combination with atropine.

(*g*) *Renal Symptoms.*—With ruptured compensation and lowering of the tension in the aorta, the urinary secretion is greatly diminished, and the amount may sink to 5 or 6 ounces in the day. Digitalis, and strophanthus when efficient, usually increase the flow. A brisk purge may be followed by augmented secretion. The combination in pill form of digitalis, squill, and the black oxide of mercury, will sometimes prove effective when the infusion or tincture of digitalis alone has failed. Calomel acts well in some cases, given in 3-grain doses every six hours for three or four days.

The *diet* in chronic valve-diseases is often very difficult to regulate. With the dilatation and venous engorgement come nausea and often a great distaste for food. The amount of liquid should be restricted, and milk, beef-juice, or egg albumen given every three hours. When the serious symptoms have passed, eggs, scraped meat, fish, and fowl may be allowed. Starchy foods, and all articles likely to cause flatulency, should be forbidden. Stimulants are usually necessary, either whisky or brandy.

III. HYPERTROPHY AND DILATATION.

Hypertrophy is an enlargement of the heart due to an increased thickness, total or partial, in the muscular walls. Dilatation is an increase in size of one or more of the chambers, with or without thickening of the walls. The conditions usually coexist, and could be more correctly described together under the term enlargement of the heart. Simple hypertrophy, in which the cavities remain of a normal size and the walls are increased, occurs, but simple dilatation, in which the cavities are increased and the walls remain of a normal diameter, probably does not, as it is always associated with thinning or with thickening of the coats. Commonly we have the forms of simple hypertrophy, hypertrophy with dilatation, and dilatation with thinning of the coats.

Hypertrophy of the Heart.

There are two forms—the simple hypertrophy, in which the cavity or cavities are of normal size; and hypertrophy with dilatation (eccentric hypertrophy), in which the cavities are enlarged and the walls increased in thickness. The condition formerly spoken of as concentric hypertrophy, in which there is diminution in the size of the cavity with thickening of the walls, is, as a rule, a post-mortem change.

The enlargement may affect the entire organ, one side, or only one chamber. Naturally, as the left ventricle does the chief work in forcing

the blood through the systemic arteries, the change is most frequently found in it.

Etiology.—Hypertrophy of the heart follows the law governing muscles, that within certain limits, if the nutrition is kept up, increased work is followed by increased size—i. e., hypertrophy. Hypertrophy of the left ventricle alone, or with general enlargement of the heart, is brought about by—

Conditions affecting the heart itself: (1) Disease of the aortic valve; (2) mitral insufficiency; (3) pericardial adhesions; (4) sclerotic myocarditis; (5) disturbed innervation, with overaction, as in exophthalmic goitre, in long-continued nervous palpitation, and as a result of the action of certain articles, such as tea, alcohol, and tobacco. In all of these conditions the work of the heart is increased. In the case of the valve lesions the increase is due to the increased intraventricular pressure; in the case of the adherent pericardium and myocarditis, to direct interference with the symmetrical and orderly contraction of the chambers.

Conditions acting upon the blood-vessels: (1) General arterio-sclerosis, with or without renal disease; (2) all states of increased arterial tension induced by the contraction of the smaller arteries under the influence of certain toxic substances, which, as Bright suggested, "by affecting the minute capillary circulation, render greater action necessary to send the blood through the distant subdivisions of the vascular system"; (3) prolonged muscular exertion, which enormously increases the blood-pressure in the arteries; (4) narrowing of the aorta, as in the congenital stenosis.

Hypertrophy of the right ventricle is met with under the following conditions—

(1) Lesions of the mitral valve, either incompetence or stenosis, which act by increasing the resistance in the pulmonary vessels. (2) Pulmonary lesions, obliteration of any number of blood-vessels within the lungs, such as occurs in emphysema or cirrhosis, is followed by hypertrophy of the right ventricle. (3) Valvular lesions on the right side occasionally cause hypertrophy in the adult, not infrequently in the fœtus. (4) Chronic valvular disease of the left heart and pericardial adhesions are sooner or later associated with hypertrophy of the right ventricle.

In the auricles simple hypertrophy is never seen; it is always dilatation with hypertrophy. In the left auricle the condition develops in lesions at the mitral orifice, particularly stenosis. The right auricle hypertrophies when there is greatly increased blood-pressure in the lesser circulation, whether due to mitral stenosis or pulmonary lesions. Narrowing of the tricuspid orifice is a less frequent cause.

Morbid Anatomy.—The heart of an average-sized man weighs about 9 ounces (280 grammes); that of a woman, about 8 ounces (250 grammes). In case of general hypertrophy the heart may weigh from 16 to 20 ounces. Weights above 25 ounces are rare. So far as I know, the heaviest heart on record is one of 53 ounces, described by Beverly Robinson. Dulles has reported one weighing 48 ounces. The measurement of the thickness of the walls is, next to weighing, the best means of determining the hypertrophy. In extreme dilatation the walls, though actually thickened, may

look thin. When *rigor mortis* is present, the cavity may be small and the walls may appear greatly thickened. The measurements should not be made until the heart has been soaked in water and thoroughly relaxed. In the left ventricle a thickness of ten lines, or from 20 to 25 mm., indicates hypertrophy. The right ventricle is thinner than the left, and has an average diameter of from 4 to 7 mm. In hypertrophy it may measure from 13 to 20 mm. The left auricle has a normal thickness of about 3 mm., which may be doubled in hypertrophy. The wall of the right auricle is thinner than that of the left, rarely exceeding 2 mm. in diameter. The appendices of the auricles often present marked increase in thickness and the musculi pectinati are greatly developed.

The shape of the heart is altered in hypertrophy; with great enlargement of the ventricles, the apex is broadened, and the conical shape is lost. In the enormous enlargement of aortic insufficiency this rotundity of the apex is very marked. When the right ventricle is chiefly affected it occupies the largest share of the apex. In mitral stenosis the contrast is very striking between the large, broad right ventricle, reaching to the apex, and the small left chamber.

The hypertrophied muscle has a deep red color, is firm, and is cut with increasing resistance. The right ventricle, as Rokitansky noted, may have a peculiar hard, leathery consistence. In simple hypertrophy of the left ventricle the papillary muscles and the columnæ carneæ may be enlarged, but the former are often much flattened in dilated hypertrophy. The muscular trabeculæ are more developed, as a rule, in the right ventricle than in the left.

The increase in size of the heart is probably due to a definite numerical increase, resulting from development of new fibres.

Symptoms.—Hypertrophy is a conservative process, secondary to some valvular or arterial lesion, and is not necessarily accompanied by symptoms. So admirable is the adjusting power of the heart that, for example, an advancing stenosis of aortic or mitral orifice may for years be perfectly equalized by a progressive hypertrophy, and the subject of the affection be happily unconscious of the existence of heart trouble. Hypertrophy is in almost all cases an unmixed good; the symptoms which arise are usually to be attributed to its failure, or, as we say, to disturbance of compensation.

Among the most common symptoms are unpleasant feelings about the heart—a sense of fulness and discomfort, rarely amounting to pain. This may be very noticeable when the patient is recumbent on the left side. Actual pain is rare, except in the irritable heart from tobacco or in neurasthenics. Palpitation may not occur, nor do patients always have sensations from the violent shocks of a greatly hypertrophied organ. There are instances in which very uneasy feelings arise from a moderately exaggerated pulsation. The general condition has much to do with this. In health we are not conscious of the heart's pulsations, but one of the first indications of exhaustion from excesses or overstudy is the consciousness of the heart's action, not necessarily with palpitation. Headaches, flushings of the face, noises in the ears, and flashes of light may be present.

Certain untoward effects of long-continued hypertrophy of the left ventricle must be mentioned, chief among which is the production of arterio-sclerosis. Particularly is this the case when the hypertrophy results from increased peripheral resistance. The heightened blood-pressure (expressed by the word strain) in the arteries gradually induces an endarteritis and a stiff, inelastic state of those vessels most exposed to it—viz., the aorta and its primary divisions. In overcoming the peripheral obstruction the hypertrophy "ruins the arteries as a sequential result" (Fothergill). Prolonged muscular exertion also acts injuriously in this way.

Another danger is rupture of the blood-vessels, particularly those of the brain. In general arterial degeneration associated with contracted kidneys and hypertrophied left heart apoplexy is common. Indeed, in the majority of cases of cerebral hæmorrhage there is sclerosis of the smaller vessels, often with the development of miliary aneurisms, and the rupture may be caused by the forcible action of the heart.

Physical Signs.—*Inspection* may show bulging of the præcordia, producing in children marked asymmetry of the chest. It may occur without pericardial adhesions, which Schroetter thinks are invariably associated with this condition. The intercostal spaces are widened, and the area of visible impulse is much increased. On *palpation* the impulse is forcible and heaving, and with each systole the hand or the ear applied over the heart may be visibly raised. A slow, heaving impulse is one of the best signs of simple hypertrophy. With large dilated hypertrophy the forcible impulse is often more sudden and abrupt. A second, weaker impulse can sometimes be felt, due perhaps to a rebound from the aortic valves (Gowers). The beat may be felt in the sixth, seventh, or eighth interspace from 1 to 3 inches outside the nipple. This downward dislocation of the apex is an important sign in hypertrophy of the left ventricle. In moderate grades, such as are seen in chronic Bright's disease, the impulse may be in the sixth interspace in the nipple line, or a little outside of it.

Percussion reveals increased dulness, which in the parasternal line may begin at the third rib or in the second interspace, and transversely may extend from half an inch to 2 inches beyond the nipple line and an equal distance beyond the middle line of the sternum. The dull area is more ovoid than in health. When carefully delimited the colossal hypertrophy of aortic valve disease may give an area of dulness from 7 to 8 inches in transverse extent. In moderate grades a transverse dulness of 4 inches is not uncommon.

On *auscultation* the sounds, when the valves are healthy, may present no special changes, but the first sound is often prolonged and dull. When there is dilatation as well, it may be very clear and sharp. Reduplication is common in the hypertrophy of renal disease. A peculiar clink—the *tintement métallique* of Bouillaud—may be heard just to the right of the apex beat. The second sound is clear and loud, sometimes ringing in character or reduplicated. With valvular lesions, the sounds, of course, are much altered, and are replaced or accompanied by murmurs.

In simple hypertrophy not dependent on valvular lesions, the pulse is usually regular, full, strong, and of high tension. It may be increased

in rapidity, but is often normal. In eccentric hypertrophy the pulse is full, but softer, and usually more rapid. One of the earliest signs of failure and dilatation is irregularity and intermittence of the pulse.

Hypertrophy of the *right ventricle* in the adult very rarely follows valvular disease on the right side, but results from increased resistance in the pulmonary circulation, as in cirrhosis of the lung and emphysema, or in stenosis of the mitral orifice. With perfect compensation, which fully maintains the equilibrium of the circulation, there are no symptoms. Extra exertion, as the ascent of stairs or running, may cause shortness of breath, but in many ways hypertrophy of the right ventricle is the most enduring and salutary form in the whole cycle of cardiac affections. For long periods of years the effects of mitral stenosis may be counterbalanced, and only sudden death by accident or an acute disease reveal the existence of an unsuspected lesion. In the hypertrophy secondary to emphysema or cirrhosis of the lungs, there may be sensations of distress in the cardiac region, with cough and shortness of breath; but as long as the dilatation is moderate the symptoms are not marked. With great dilatation and tricuspid leakage come venous engorgement, œdema, and pulmonary troubles. The increased pressure in the lesser circulation leads to sclerosis of the pulmonary arteries and the constant engorgement of the capillaries leads ultimately to a deposition of pigment and increase in the fibrous elements in the lung—the brown induration. Extreme pulmonary congestion and apoplexy are more often associated with dilatation. Hæmoptysis may result from rupture of vessels during sudden exertion.

Physical Signs.—Bulging of the lower part of the sternum and left cartilages occurs. The apex beat is forced to the left, but is not so often displaced downward. The most marked impulse may be in the angle between the ensiform cartilage and the seventh rib or beneath the cartilages of the sixth and seventh ribs. The pulsation is rather diffuse, not punctate, particularly if there is much dilatation. In thin-walled chests there may be pulsation in the third and fourth right interspaces. The cardiac dulness is increased transversely and toward the right; it may extend an inch or more beyond the border of the sternum. On auscultation the first sound at the lower part of the sternum is louder and fuller than normal, but the differences are not very marked unless there is much dilatation, when the sound is clearer and sharper. Accentuation and reduplication of the second sound are heard in the pulmonary artery on account of the increased tension. The pulse at the wrist is usually small. Pulsation occurs in the jugulars when there is tricuspid incompetence.

Hypertrophy of the *auricles* always occurs with dilatation. It is more common in the left chamber, which hypertrophies in mitral stenosis and incompetency, and naturally assists in restoring the balance of the circulation. There are no distinctive physical signs, and we usually can infer its presence only by the existence of mitral stenosis and a presystolic murmur. Increased dulness may be determined to the left of the sternum, and there may be a presystolic wave in the second left interspace.

Hypertrophy and dilatation of the right auricle are met with (associated with a similar condition in the right ventricle and incompetency of

the tricuspid) in emphysema, cirrhosis of the lung, chronic bronchitis, and mitral disease. In comparison with the left auricle the greater development and hypertrophy of the appendix and its musculi pectinati is very striking. The latter may be distributed over the anterior wall of the sinus to a greater extent than in health. There are increased dulness in the third and fourth interspaces, pulsation sometimes presystolic in rhythm, signs of venous engorgement, jugular pulsation, and other evidences of dilatation of the right heart.

Diagnosis.—Among conditions to be distinguished are:

(1) Neurotic palpitation, from whatever cause, even when very forcible, has not the heaving impulse of genuine hypertrophy. Enlargement of the organ may, however, follow prolonged overaction, as in the smoker's heart, the irritable heart of neurasthenics, and in exophthalmic goitre, but it is usually slight.

(2) The increased area of dulness may be due to a variety of causes, some of which may closely simulate hypertrophy, such as pericardial effusion, aneurism, mediastinal growths, or displacement of the heart from pressure, or the existence of malformation of the chest. With the exercise of ordinary care, however, the diagnosis can usually be made. There are two opposite conditions which frequently give trouble. With the left lung contracted from pleurisy, phthisis, or cirrhosis, a large surface of the heart is exposed; the pulsation may be extensive and forcible, and may at first sight suggest hypertrophy. In this condition there is dislocation upward and to the left. The existence of pulmonary or pleuritic disease and the fixation of the lung on deep inspiration will suffice to prevent mistakes. A less extensive exposure of the heart may occur without any disease in very narrow-chested persons with ill-developed lungs; here, though the area of dulness may be much increased, the normal position of the apex, the absence of forcible, heaving impulse, and of any obvious cause of hypertrophy will afford satisfactory criteria for a diagnosis. The reverse condition exists in some cases in which emphysema masks moderate cardiac hypertrophy. The area of dulness may be normal, or even diminished, and the pulse and character of the sounds will help in the diagnosis; but it is sometimes a difficult matter.

Prognosis.—The course of any case of cardiac hypertrophy may be divided into three stages:

(*a*) The period of development, which varies with the nature of the primary lesion. For example, in rupture of an aortic valve, during a sudden exertion, it may require months before the hypertrophy becomes fully developed; or, indeed, it may never do so, and death may follow from an uncompensated dilatation. On the other hand, in sclerotic affections of the valves, with stenosis or incompetency, the hypertrophy develops step by step with the lesion, and may continue to counterbalance the progressive and increasing impairment of the valve.

(*b*) The period of full compensation—the latent stage—during which the heart's vigor meets the requirements of the circulation. This period may last an indefinite time, and a patient may never be made aware by any symptoms that he has a valvular lesion.

(*c*) The period of broken compensation, which may come on suddenly during very severe exertion. Death may result from acute dilatation; but more commonly it takes place slowly and results from degeneration and weakening of the heart-muscle.

The breaking or rupture of cardiac compensation may be induced by many causes, among which the most important are: (1) Failure of the general nutrition. In many instances of heart-disease, exposure, poor food, and alcohol combine to bring about disturbance of a well-balanced heart lesion. Acute illnesses, particularly the fevers, may induce general debility and with it weakening of the heart-muscle. (2) Disturbance of the local nutrition of the heart, owing to gradual sclerosis of the coronary arteries, is a common cause. (3) Very severe muscular exertion, which may disturb a compensation, perfect for years, and induce death in a few days (Traube). (4) Mental emotions. Severe grief or fright may bring on failure of compensation.

The prognosis is largely, as already stated, a matter of maintained compensation. Once established, the hypertrophy rarely, if ever, disappears, inasmuch as the cause usually persists. Occasionally, perhaps, the hypertrophy associated with neurotic palpitation from tobacco, or other causes, or the hypertrophy following muscular overexertion, may disappear.

Dilatation of the Heart.

Two varieties are recognized, dilatation with thickening and dilatation with thinning. The former is the more common, and corresponds to the dilated or eccentric hypertrophy.

Etiology.—Two important causes combine to produce dilatation—increased pressure within the cavities and impaired resistance, due to weakening of the muscular wall—which may act singly, but are often combined. A weakened wall may yield to a normal distending force, or a normal wall may yield under a heightened blood-pressure.

(1) Heightened endocardiac pressure results either from an increased quantity of blood to be moved or an obstacle to be overcome, and is the more frequent cause. It does not necessarily bring about dilatation; simple hypertrophy may follow, as in the early period of aortic stenosis, and in the hypertrophy of the left ventricle in Bright's disease.

A majority of the important causes of increased endocardiac pressure have already been discussed under hypertrophy. One or two may be considered more in detail.

The size of the cardiac chambers varies in health. With slow action of the heart the dilatation is complete and fuller than it is with rapid action. Physiologically, the limits of dilatation are reached when the chamber does not empty itself during the systole. This may occur as an acute, transient condition in severe exertion—during, for example, the ascent of a mountain. There may be great dilatation of the right heart, as shown by the increased epigastric pulsation, and even increase in the cardiac dulness. The safety-valve action of the tricuspid valves may here come into play, relieving the lungs by permitting regurgitation into the

auricle. With rest the condition is removed, but if it has been extreme, the heart may suffer a strain from which it may recover slowly, or, indeed, the individual may never be able again to undertake severe exertion. In the process of training, the getting wind, as it is called, is largely a gradual increase in the capability of the heart, particularly of the right chambers. A degree of exertion can be safely maintained in full training which would be quite impossible under other circumstances, because, by a gradual process of what we may call physical education, the heart has strengthened its reserve force—widened enormously its limits of physiological work. Endurance in prolonged contests is measured by the capabilities of the heart, and its essence consists in being able to meet the continuous tendency to overstep the limits of dilatation.

We have no positive knowledge of the nature of the changes in the heart which occur in this process, but it must be in the direction of increased muscular and nervous energy. The large heart of athletes may be due to the prolonged use of their muscles, but no man becomes a great runner or oarsman who has not naturally a capable if not a large heart. Master McGrath, the celebrated greyhound, and Eclipse, the race-horse, both famous for endurance rather than speed, had very large hearts.

Excessive dilatation during severe muscular effort results in heart-strain. A man, perhaps in poor condition, calls upon his heart for extra work during the ascent of a high mountain, and is at once seized with pain about the heart and a sense of distress in the epigastrium. He breathes rapidly for some time, is "puffed," as we say, but the symptoms pass off after a night's quiet. An attempt to repeat the exercise is followed by another attack, or, indeed, an attack of cardiac dyspnœa may come on while he is at rest. For months such a man may be unfitted for severe exertion, or he may be permanently incapacitated. In some way he has overstrained his heart and become "broken-winded." Exactly what has taken place in these hearts we cannot say, but their reserve force is lost, and with it the power of meeting the demands exacted in maintaining the circulation during severe exertion. The "heart-shock" of Latham includes cases of this nature—sudden cardiac breakdown during exertion, not due to rupture of a valve. It seems probable that sudden death in men during long-continued efforts, as in a race, is sometimes due to overdistention and paralysis of the heart.

Examples of dilatation occur in all forms of valve lesions. In aortic incompetency blood enters the left ventricle during diastole from the unguarded aorta and from the left auricle, and the quantity of blood at the termination of diastole subjects the walls to an extreme degree of pressure, under which they inevitably yield. In time they augment in thickness, and present the typical eccentric hypertrophy of this condition.

In mitral insufficiency blood which should have been driven into the aorta is forced into and dilates the auricle from which it came, and then in the diastole of the ventricle a large amount is returned from the auricle, and with increased force. In mitral stenosis the left auricle is the seat of greatly increased tension during diastole, and dilates as well as hypertrophies; the distention, too, may be enormous. Dilatation of the

right ventricle is produced by a number of conditions, which were considered under hypertrophy. All circumstances, such as mitral stenosis, emphysema, etc., which permanently increase the tension of the blood in the pulmonary vessels, cause its dilatation.

(2) Impaired nutrition of the heart-walls may lead to a diminution of the resisting power so that dilatation readily occurs.

The loss of tone due to parenchymatous degeneration or myocarditis in fevers may lead to a fatal condition of acute dilatation. It is a recognized cause of death in scarlatinal dropsy (Goodhart), and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. The changes in the heart-muscle which accompany acute endocarditis or pericarditis may lead to dilatation, especially in the latter disease. In anæmia, leukæmia, and chlorosis the dilatation may be considerable. In sclerosis of the walls, the yielding is always where this process is most advanced, as at the left apex. Under any of these circumstances the walls may yield with normal blood-pressure.

Pericardial adhesions are a cause of dilatation, and we generally find in cases with extensive and firm union considerable hypertrophy and dilatation. There is usually here some impairment as well of the superficial layers of muscle.

Morbid Anatomy.—The condition usually exists with hypertrophy in two or more chambers. It is more common on the right than on the left side. The most extreme dilatation is in cases of aortic incompetency, in which all the cavities may be enormously distended. In mitral stenosis the left auricle is often trebled in capacity, and the right chambers also are very capacious. The auricles may contain from 18 to 20 ounces of blood. In chronic lesions of the lungs the right chambers are chiefly involved. In great distention of one ventricle the septum may bulge toward the other side. The auriculo-ventricular rings are often dilated, and there may be an increase in the circumference of 1½ or even 2 inches. Thus, the tricuspid orifice, the circumference of which is about 4½ inches, may freely admit a graduated heart-cone of above 6 inches; and the mitral orifice, which normally is about 3½ inches, may admit the cone to 5½ inches or even more. Great dilatation is always accompanied by relative incompetency of the valves, so that free regurgitation into the auricles is permitted. The orifices of the venæ cavæ and of the pulmonary veins may be greatly dilated.

The endocardium is often opaque, particularly that of the auricles. The muscle substance varies according to the presence or absence of degenerations. The microscope may show marked fatty or parenchymatous change, but in some instances no special alteration may be noticeable. There is much truth in Niemeyer's assertion "that it is not possible by means of the microscope to recognize all the alterations of the muscular fibrillæ which diminish the functional power of the heart." Of the changes in the ganglia of the heart we know very little. As centres of control they probably have more to do with cardiac atony and breakdown than we generally admit. Degeneration of them has been noted by Putjakin, Ott, and others.

Symptoms and Physical Signs.—Dilatation causes weakness of the cardiac walls, diminishes the vigor of their contractions, and is therefore the reverse of hypertrophy. So long as compensation is maintained the enlargement of a cavity may be considerable. The limit is reached when the hypertrophied walls in the systole can no longer expel all the contents, part of which remain, so that at each diastole the chamber is abnormally full. Thus, in aortic incompetency blood enters the left ventricle from the aorta as well as the auricle; dilatation ensues, and also hypertrophy as a direct effect of the increased pressure and increased amount of blood to be moved. But if from any cause the hypertrophy weakens and the ventricle during systole fails to empty itself completely, a still larger amount is in it at the end of each diastole, and the dilatation becomes greater. The amount remaining after systole prevents the blood from entering freely from the auricle. Incompetency of the auriculo-ventricular valves follows, with dilatation of the auricle and impeded blood-flow in the pulmonary veins. Dilatation and hypertrophy of the right heart may compensate for a time, but when this fails the venous system becomes engorged and dropsy may result. The consideration of the symptoms of chronic valvular lesions is largely that of dilatation and its effects. Acute dilatation, such as we see in fevers or in sudden failure of a hypertrophied heart, is accompanied by three chief symptoms—weak, usually rapid, impulse, dyspnœa, and signs of obstructed venous circulation. Cardiac pain may be present, but is often absent.

The *physical signs* of dilatation are those of a weak and enlarged organ. The impulse is diffuse, often undulatory, and is felt over a wide area, and an apex beat or a point of maximum intensity may not be found. When it does exist, it may be visible and yet cannot be felt—a valuable observation made by Walshe. An extensive area of impulse with a quick, weak maximum apex beat may be present. When the right heart is chiefly dilated the left may be pushed over so as to occupy a much less extensive area in front of the heart, and the true apex beat cannot be felt; but the chief impulse is just below, or to the right of, the xiphoid cartilage, and there is a wavy pulsation in the fourth, fifth, and sixth interspaces to the left of the sternum. In extreme dilatation of the right auricle a pulsation may sometimes be seen in the third right interspace close to the sternum, and with free tricuspid regurgitation this may be systolic in character. Whether the pulsation frequently seen in the second left interspace is ever due to a dilated left auricle has not been determined. I have sometimes thought it was presystolic in rhythm, though it may be distinctly systolic. Post mortem, it is rare in the most extreme distention to see the auricular appendix so far forward as to warrant the belief that it could beat against the second interspace. The area of dulness is increased, but an emphysematous lung or the fully distended organ in a state of brown induration may cover over the heart and greatly limit the extent. The directions of increase were considered in connection with hypertrophy.

The first sound is shorter, sharper, more valvular in character, and more like the second. As the dilatation becomes excessive it gets weaker. Reduplication is not common, but occasionally differences may be heard

in the first sound over the right and left hearts. The sounds are frequently obscured by murmurs, which are produced by incompetency of the valves due to the great dilatation, or are associated with the chronic valve disease on which the condition depends. The aortic second sound is replaced by a murmur in aortic regurgitation. The pulmonary sound is accentuated in mitral regurgitation and pulmonary congestion, but with extreme dilatation it may be much weakened. The heart's action is irregular and intermittent, and the pulse is small, weak, and quick.

On auscultation both the sounds may be free from murmur. There is the condition known as embryocardia or fœtal heart-rhythm, in which the first and second sounds are very alike, and the long pause is shortened. In other instances there is the typical and characteristic gallop rhythm, rarely found apart from conditions of dilatation. With the various valvular lesions the corresponding murmurs may be heard. Murmurs, however, which have been present may disappear, as in the case of mitral stenosis. In other instances a loud systolic murmur may be heard at the apex, and when the case first comes under observation it may be impossible to say whether this is due to organic mitral lesion. The murmur may be confined to the apex region, or propagated well to the back. It is extremely common in the dilatation which follows the hypertrophy of the left ventricle in arterio-sclerosis. Under treatment, with the gradual disappearance of the dilatation, a murmur of this kind, even though most intense, may completely disappear, showing that it has been due to a relative insufficiency, not to a valvular lesion. All varieties of arrhythmia may occur in dilatation of the heart. The pulse, as a rule, is small, weak, quick, and often irregular.

Dilation and Hypertrophy due to Overexertion and Alcohol.—There is a group of cases of dilatation and hypertrophy dependent upon prolonged overexertion, which rarely comes under observation until compensation has failed, and which then may be very difficult to distinguish from the similar conditions produced by valvular disease. The patients are able-bodied men at the middle period of life, and complain first of palpitation or irregularity of the action of the heart and shortness of breath; subsequently the usual symptoms of cardiac insufficiency develop. On inquiring into the history of these patients none of the usual etiological factors causing valve-disease are present, but they have always been engaged in laborious occupations and have usually been in the habit of taking stimulants freely. This is the affection which has been specially studied by McLean, Clifford Albutt, Seitz, and others, and in its earlier condition by Da Costa, in what he termed the irritable heart. It is met with very frequently in soldiers. These cases may return to hospital three or four times with cardiac insufficiency, sometimes with slight anasarca, hæmoptysis, and signs of pulmonary engorgement. The condition is by no means infrequent. Bollinger has called attention to the common occurrence of dilatation and hypertrophy in beer-drinkers, particularly in the workers in the German breweries, who drink 20 or more litres in the day. Strümpell, at his Erlangen clinic, told me that this condition was very common in the draymen and workers in the breweries of that town, very few of

whom pass the forty-fifth year without indications of hypertrophy and dilatation of the heart. On post-mortem examination the valves may be quite healthy, the aorta smooth, and extensive arterio-sclerosis or renal disease absent. The heart weighs from 18 to 25 ounces; the chambers are dilated. The condition has been met with also in animals, and Houghton states that the heart of the celebrated greyhound Master McGrath weighed 9.57 ounces, just threefold in excess of the normal proportion of heart-weight to body-weight.

Idiopathic Dilatation.—And, lastly, there are other cases in which dilatation of the heart occurs without discoverable cause. In some instances there has been a history of sudden exercise or of mental emotion, but in other cases the condition seems to have come on spontaneously. In some it is acute and the patient has dyspnœa, slight cyanosis, cough, and great cardiac distress. Death may occur in a few days, or dropsy may supervene and the case may become chronic. Delafield has reported an interesting series of cases of this group.

Treatment.—The treatment of hypertrophy and dilatation has already been considered under the section on valvular lesions. I would only here emphasize the fact that with signs of dilatation, as indicated by gallop rhythm, urgent dyspnœa, and slight lividity, venesection is in many cases the only means by which the life of the patient may be saved, and from 25 to 30 ounces of blood should be abstracted without delay. Subsequently stimulants, such as ammonia and digitalis, may be administered, but they are accessories only to the bleeding in the critical condition of acute dilatation, which is so frequently met with in cardiac lesions.

IV. AFFECTIONS OF THE MYOCARDIUM.

1. **Lesions due to Disease of the Coronary Arteries.**—A knowledge of the changes produced in the myocardium by disease of the coronary vessels gives a key to the understanding of many problems in cardiac pathology. The terminal branches of the coronary vessels are end-arteries; that is, the communication between neighboring branches is through capillaries only. F. H. Pratt * has lately shown that the vessels of Thebesius, which open from the ventricles and auricles into a system of fine branches and thus communicate with the cardiac capillaries and coronary veins, may be capable of feeding the myocardium sufficiently to keep it alive even when the coronary arteries are occluded. The blocking of one of these vessels by a thrombus or an embolus leads usually to a condition which is known as—

(*a*) *Anæmic necrosis*, or white infarct. When this does not occur the reason may be sought in (1) the existence of abnormal anastomoses, which by their presence take the coronary system out of the group of end-arteries; or (2) the vicarious flow through the vessels of Thebesius and the coronary veins. The condition is most commonly seen in the left ventricle and in the septum, in the territory of distribution of the anterior coronary artery. The

* The American Journal of Physiology, vol. i, 1898.

affected area has a yellowish-white color, sometimes a turbid, parboiled aspect, at other times a grayish-red tint. It may be somewhat wedge-shaped, more often it is irregular in contour and projects above the surface. Microscopically the changes are very characteristic. The nuclei either disappear from the muscle fibres or they undergo fragmentation. Leucocytes wander in from the surrounding tissue, and these may suffer disintegration. At a later stage a new growth of fibrous tissue is found in the periphery of the infarct which ultimately may entirely replace the dead fibres. The fibres present a homogeneous, hyaline appearance. In some instances there is complete transformation, and even to the naked eye a firm white patch of hyaline degeneration may appear in the centre of the area. Sudden death not infrequently follows the blocking of one of the branches of the coronary artery and the production of this anæmic necrosis. *In medico-legal cases it is a point of primary importance to remember that this is one of the common causes of sudden death.* This condition should be carefully sought for, inasmuch as it may be the sole lesion, except a general, sometimes slight arterio-sclerosis. Rupture of the heart may be associated with anæmic necrosis.

(*b*) The second important effect of coronary-artery disease upon the myocardium is seen in the production of *fibrous myocarditis.* This may result from the gradual transformation of areas of anæmic necrosis. More commonly it is caused by the narrowing of a coronary branch in a process of obliterative endarteritis. Where the process is gradual evidences of granulation tissue are often wanting, and any distinction between the necrotic muscle fibres and the new scar tissue is difficult to establish. J. B. MacCallum has shown that the muscle fibres undergo a change the reverse of that of their normal development and lose their fibril bundles preliminary to their complete replacement by connective tissue. The sclerosis is most frequently seen at the apex of the left ventricle and in the septum, but it may occur in any portion. In the septum and walls there are often streaks and patches which are only seen in carefully made systematic sections. Hypertrophy of the heart is commonly associated with this degeneration. It is the invariable precursor of aneurism of the heart.

Complete obliteration of one coronary artery, if produced suddenly, is usually fatal. When induced slowly, either by arterio-sclerosis at the orifice of the artery at the root of the aorta or by an obliterating endarteritis in the course of the vessel, the circulation may be carried on through the other vessel. Sudden death is not uncommon, owing to thrombosis of a vessel which has become narrowed by sclerosis. In the most extreme grade one coronary artery may be entirely blocked, with the production of extensive fibroid disease, and a main branch of the other also may be occluded. A large, powerfully built imbecile, aged thirty-five, at the Elwyn Institution, Pennsylvania, who had for years enjoyed doing the heavy work about the place, died suddenly, without any preliminary symptoms. The heart, which is in my collection, weighed over 20 ounces; the anterior coronary artery was practically occluded by obliterating endarteritis, and of the posterior artery one main branch was blocked.

(*c*) *Septic Infarcts.*—In pyæmia the smaller branches of the coronary

arteries may be blocked with emboli which give rise to infectious or septic infarcts in the myocardium in the form of abscesses, varying in size from a pea to a pin's head. These may not cause any disturbance, but when large they may perforate into the ventricle or into the pericardium, forming what has been called acute ulcer of the heart.

2. **Acute Interstitial Myocarditis.**—In some infectious diseases and in acute pericarditis the intermuscular connective tissue may be swollen and infiltrated with small round cells and leucocytes, the blood-vessels dilated, and the muscle fibres the seat of granular, fatty, and hyaline degeneration. Occasionally, in pyæmia the infiltration with pus-cells has been diffuse and confined chiefly to the interstitial tissue. Councilman has described this condition of the heart wall in gonorrhœa, and succeeded in demonstrating the gonococcus in the diseased areas. The commonest examples are found in diphtheria, typhoid fever, and acute endocarditis, as shown by the studies of Romberg. The foci may be the starting-points of patches of fibrous myocarditis.

3. **Fragmentation and Segmentation.**—This condition was described by Renaut and Landouzy in 1877, and has been carefully studied by different pathologists.* Two forms are met with: 1. Segmentation. The muscle fibres have separated at the cement line. 2. Fragmentation. The fracture has been across the fibre itself, and perhaps at the level of the nucleus. Longitudinal division is unusual. Although the condition doubtless arises in some instances during the death agony, as in cases of sudden death by violence, in others it would seem to have clinical and pathological significance. It is found associated with other lesions, fibrous myocarditis, infarction, and fatty degeneration. J. B. MacCallum distinguishes a simple from a degenerative fragmentation. The first takes place in the normal fibre, which, however, shows irregular extensions and contractions. The second succeeds degeneration in the fibre. Hearts the seat of marked fragmentation are lax, easily torn, the muscle fibres widely separated, and often pale and cloudy.

4. **Parenchymatous Degeneration.**—This is usually met with in fevers, or in connection with endocarditis or pericarditis, and in infections and intoxications generally. It is characterized by a pale, turbid state of the cardiac muscle, which is general, not localized. Turbidity and softness are the special features. It is the softened heart of Laennec and Louis. Stokes speaks of an instance in which "so great was the softening of the organ that when the heart was grasped by the great vessels and held with the apex pointing upward, it fell down over the hand, covering it like a cap of a large mushroom."

Histologically, there is a degeneration of the muscle fibres, which are infiltrated to a various extent with granules which resist the action of ether, but are dissolved in acetic acid. Sometimes this granular change in the fibres is extreme, and no trace of the striæ can be detected. It is probably the effect of a toxic agent, and is seen in its most exquisite form in the lumbar muscles in cases of toxic hæmoglobinuria in the horse. It is met

* Hektoen, American Journal of the Medical Sciences, 1897.

with in cases of typhoid, typhus, small-pox, and other infectious diseases, particularly when the course is protracted. There is no definite relation between it and the high temperature.

5. **Fatty Heart.**—Under this term are embraced fatty degeneration and fatty overgrowth.

(*a*) *Fatty degeneration* is a very common condition, and mild grades are met with in many diseases. It is found in the failing nutrition of old age, of wasting diseases, and of cachectic states; in prolonged infectious fevers, in which it may follow or accompany the parenchymatous change; associated with acute and chronic anæmias. Certain poisons, such as phosphorus, produce an intense fatty degeneration. Local causes: Pericarditis is usually associated with fatty or parenchymatous changes in the superficial layers of the myocardium. Disease of the coronary arteries is a common and important cause, and it is associated with fat embolism. Lastly, in the hypertrophied ventricular wall in chronic heart-disease fatty change is by no means infrequent. This degeneration may be limited to the heart or it may be more or less general in the solid viscera. The diaphragm may also be involved, even when the other muscles show no special changes. There appears to be a special proneness to fatty degeneration in the heart-muscle, which may perhaps be connected with its incessant activity. So great is its need of an abundant oxygen supply that it feels at once any deficiency, and is in consequence the first muscle to show nutritional changes.

Anatomically the condition may be local or general. The left ventricle is most frequently affected. If the process is advanced and general, the heart looks large and is flabby and relaxed. It has a light yellowish-brown tint, or, as it is called, a faded-leaf color. Its consistence is reduced and the substance tears easily. In the left ventricle the papillary columns and the muscle beneath the endocardium show a streaked or patchy appearance. Microscopically, the fibres are seen to be occupied by minute globules distributed in rows along the line of the primitive fibres (Welch). In advanced grades the fibres seem completely occupied by the minute globules.

(*b*) *Fatty Overgrowth.*—This is usually a simple excess of the normal subpericardial fat, to which the term *cor adiposum* was given by the older writers. In pronounced instances the fat infiltrates between the muscular substance and, separating the strands, may reach even to the endocardium. In corpulent persons there is always much pericardial fat. It forms part of the general obesity, and occasionally leads to dangerous or even fatal impairment of the contractile power of the heart. Of 122 cases analyzed by Forchheimer there were 88 males and 34 females. Over 80 per cent occurred between the fortieth and seventieth years.

The entire heart may be enveloped in a thick sheeting of fat through which not a trace of muscle substance can be seen. On section, the fat infiltrates the muscle, separating the fibres, and in extreme cases—particularly in the right ventricle—reaches the endocardium. In some places there may be even complete substitution of fat for the muscle substance. In rare instances the fat may be in the papillary muscles. The heart is usually much relaxed and the chambers are dilated. Microscopically the muscle fibres may show, in addition to the atrophy, marked fatty degeneration.

6. **Other Degenerations of the Myocardium.** (*a*) *Brown Atrophy.*—This is a common change in the heart-muscle, particularly in chronic valvular lesions and in the senile heart. When advanced, the color of the muscles is a dark red-brown, and the consistence is usually increased. The fibres present an accumulation of yellow-brown pigment chiefly about the nuclei. The cement substance is often unusually distinct, but seems more fragile than in healthy muscle.

(*b*) Amyloid degeneration of the heart is occasionally seen. It occurs in the intermuscular connective tissue and in the blood-vessels, not in the fibres.

(*c*) The hyaline transformation of Zenker is sometimes met with in prolonged fevers. The affected fibres are swollen, homogeneous, translucent, and the striæ are very faint or entirely absent.

(*d*) Calcareous degeneration may occur in the myocardium, and the muscle fibres may be infiltrated and yet retain their appearance as figured and described by Coats in his Text-book of Pathology.

Symptoms of Myocardial Disease.—These are notoriously uncertain. A man with advanced fibroid myocarditis may drop dead suddenly, while doing heavy work, without having complained of cardiac distress. On the other hand, a patient may present enfeebled, irregular action and signs of dilatation; he may have shortness of breath, œdema, and the general symptoms believed to be characteristic of cases of fibroid and fatty heart, and the post mortem show little or no change in the myocardium.

Cardio-sclerosis or fibroid heart is in some cases characterized by a feeble, irregular, slow pulse, with dyspnœa on exertion and occasional attacks of angina. Irregularity is present in many, but not in all cases. The pulse may be very slow, even 30 or 40 per minute. Ultimately the cases come under observation with the symptoms of cardiac insufficiency. The arrhythmia, which may have been present, becomes aggravated and, according to Riegel, may not only precede, but also persist after the cardiac insufficiency has passed away.

Fatty degeneration of the heart presents the same difficulties. Extreme fatty changes, as in pernicious anæmia, may be consistent with a full, regular pulse and a regularly acting heart. In some of these cases the fat does not appear to interfere seriously with the function of the organ. The truth is, it may exist in an extreme grade without producing symptoms, so long as great dilatation of the chambers does not occur. The cardiac irregularity, the dyspnœa, palpitation, and small pulse are in reality not symptoms of the fatty degeneration, but of dilatation which has supervened. The fatty *arcus senilis* is of no moment in the diagnosis of fatty heart. The heart-sounds may be weak and the action irregular. When dilatation occurs, there is often the gallop rhythm, shortening of the long pause, and a systolic murmur at the apex. Shortness of breath on exertion is an early feature in many cases, and anginal attacks may occur. There is sometimes a tendency to syncope, and in both fibroid and fatty heart there are attacks in which the patient feels cold and depressed and the pulse sinks to 40 or 30, or even, as in one case which I saw, to 26. The patient may wake from sleep in the early morning with an attack of severe cardiac

asthma. These "spells" may be associated with nausea and may alternate with others in which there are anginal symptoms. These are the cases, too, in which for weeks there may be mental symptoms. The patient has delusions and may even become maniacal. Toward the close, the type of breathing known as Cheyne-Stokes may occur. It was described in the following terms by John Cheyne, speaking of a case of fatty heart (Dublin Hospital Reports, vol. ii, p. 221, 1818): "For several days his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again: this revolution in the state of his breathing lasted about a minute, during which there were about thirty acts of respiration." It is seen much more frequently in arterio-sclerosis and uræmic states than in fatty heart.

Fatty overgrowth of the heart is a condition certain to exist in very obese persons. It produces no symptoms until the muscular fibre is so weakened that dilatation occurs. These patients may for years present a feeble but regular pulse; the heart-sounds are weak and muffled, and a murmur may be heard at the apex. Attacks of cardiac asthma are not uncommon, and the patient may suffer from bronchitis. Dizziness and pseudo-apoplectic seizures may occur. Sudden death may result from syncope or from rupture of the heart. The physical examination is often difficult because of the great increase in the fat, and it may be impossible to define the area of dulness.

For practical purposes we may group the cases of myocardial disease as follows:

(1) Those in which sudden death occurs with or without previous indications of heart-trouble. Sclerosis of the coronary arteries exists—in some instances with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. Many patients never complain of cardiac distress, but, as in the case of Chalmers, the celebrated Scottish divine, enjoy unusual vigor of mind and body.

(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms. These are the cases in which the condition may be strongly suspected and, in some instances, diagnosed. It is rarely possible to make a distinction between the fatty and fibroid heart.

(3) Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart. Dropsy is often present, and with a loud murmur at the apex it may be difficult, unless the case has been seen from the outset, to determine whether or not a valvular lesion is present.

Prognosis.—The outlook in affections of the myocardium is extremely grave. Patients recover, however, in a surprising way from the most serious attacks, particularly those of the second group.

Treatment.—Many cases never come under treatment; the first are the final symptoms.

Cases with signs of well-marked cardiac insufficiency, as manifested by dyspnœa, weak, irregular, rapid heart, and œdema, may be treated on the

plan laid down for the treatment of broken compensation in valvular disease. Digitalis may be given even if fatty degeneration is suspected, and is often very beneficial.

Much more difficult is the management of those cases in which there is marked cardiac arrhythmia, with a feeble, irregular, very slow pulse, and syncope or angina. Dropsy is not, as a rule, present; the heart-sounds may be perfectly clear, and there are no signs of dilatation. Digitalis, under these circumstances, is not advisable, particularly when the pulse is infrequent. Complete rest in bed, a carefully regulated diet, and the use of the aromatic spirits of ammonia, sulphuric ether, and stimulants are indicated. For the restlessness and distressing feelings of anxiety morphia is invaluable. From an eightieth to a sixtieth of a grain of strychnia may be given three times a day. If, as is sometimes the case, the pulse is hard and firm, nitroglycerin may be cautiously administered, beginning with 1 minim of the 1-per-cent solution three times a day and increased gradually.

In certain cases of weak heart, particularly when it is due to fatty overgrowth, the plans recommended by Oertel and by Schott are advantageous. They are invaluable methods in those forms of heart-weakness due to intemperance in eating and drinking and defective bodily exercise. The Oertel plan consists of three parts: First, the reduction in the amount of liquid. This is an important factor in reducing the fat in these patients. It also slightly increases the density of the blood. Oertel allows daily about 36 ounces of liquid, which includes the amount taken with the solid food. Free perspiration is promoted by bathing (if advisable, the Turkish bath), or even by the use of pilocarpine.

The second important point in his treatment is the diet, which should consist largely of proteids.

Morning.—Cup of coffee or tea, with a little milk, about 6 ounces altogether. Bread, 3 ounces.

Noon.—Three to 4 ounces of soup, 7 to 8 ounces of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; 1 ounce of bread or farinaceous pudding; 3 to 6 ounces of fruit for dessert. No liquids at this meal, as a rule, but in hot weather 6 ounces of light wine may be taken.

Afternoon.—Six ounces of coffee or tea, with as much water. As an indulgence an ounce of bread.

Evening.—One or 2 soft-boiled eggs, an ounce of bread, perhaps a small slice of cheese, salad, and fruit; 6 to 8 ounces of wine with 4 or 5 ounces of water (Yeo).

The most important element of all is graduated exercise, not on the level, but up hills of various grades. The distance walked each day is marked off and is gradually lengthened. In this way the heart is systematically exercised and strengthened.

The Schott Treatment.—This consists in a combination of baths with exercises at Nauheim. The water has a temperature of from 82°–95° F., and is very richly charged with CO_2. The good effects of the bath are claimed by Schott to come from a cutaneous excitation, induced by the mineral and gaseous constituents of the bath, and a stimulation of the sensory nerves. There is no question that the bath, in suitable cases, will

alter the position of the apex beat, and that it lessens the area of cardiac dulness; this means that it diminishes the dilatation of the heart. Artificial baths are used, consisting of forty gallons of water, with various strengths of sodium chloride and calcium chloride. The exercises, resistance gymnastics, consist in slow movements executed by the patient and resisted by the operator. Any one wishing to carry out in private the Schott treatment should consult the work of Besley Thorne. Camac's articles (J. H. H. Bulletin, vol. viii, and Jour. of the Am. Med. Assoc., 1897, ii) give a brief account of our experience with it.

Aneurism of the Heart.

(*a*) **Aneurism of a valve** results from acute endocarditis, which produces softening or erosion and may lead either to perforation of the segment or to gradual dilatation of a limited area under the influence of the blood-pressure. The aneurisms are usually spheroidal and project from the ventricular face of a sigmoid valve. They are much less common on the mitral segments. They frequently rupture and produce extensive destruction and incompetency of the valves.

(*b*) **Aneurism of the walls** results from the weakening induced by chronic myocarditis, or occasionally it follows acute mural endocarditis, which more commonly, however, leads to perforation. It has followed a stab-wound, a gumma of the ventricle, and, according to some authors, pericardial adhesions. The left ventricle near the apex is usually the seat, this being the situation in which fibrous degeneration is most common. Fifty-nine of the 60 cases collected by Legg were situated here. In the early stages the anterior wall of the ventricle, near the septum, sometimes even the septum itself, is slightly dilated, the endocardium opaque, and the muscular tissue sclerotic. In a more advanced stage the dilatation is pronounced and layers of thrombi occupy the sac. Ultimately a large rounded tumor may project from the ventricle and may attain a size equal to that of the heart. Occasionally the aneurism is sacculated and communicates with the ventricle through a very small orifice. The sac may be double, as in the cases of Janeway and Sailer. In the museum of Guy's Hospital there is a specimen showing the wall of the ventricle covered with aneurismal bulgings. Rupture occurred in 7 of the 90 cases collected by Legg.

The *symptoms* produced by aneurism of the heart are indefinite. Occasionally there is marked bulging in the apex region and the tumor may perforate the chest wall. In mitral stenosis the right ventricle may bulge and produce a visible pulsating tumor below the left costal border, which I have known to be mistaken for cardiac aneurism. When the sac is large and produces pressure upon the heart itself, there may be a marked disproportion between the strong cardiac impulse and the feeble pulsation in the peripheral arteries.

Rupture of the Heart.

This rare event is usually associated with fatty infiltration or degeneration of the heart-muscles. In some instances, acute softening in conse-

quence of embolism of a branch of the coronary artery, suppurative myocarditis, or a gummatous growth has been the cause. Of 100 cases collected by Quain, fatty degeneration was noted in 77. Two thirds of the patients were over sixty years of age.

The rent may occur in any of the chambers, but is found most frequently in the left ventricle on the anterior wall, not far from the septum. The accident usually takes place during exertion. There may be no preliminary symptoms, but without any warning the patient may fall and die in a few moments. Sudden death occurred in 71 per cent of Quain's cases. In other instances there may be in the cardiac region a sense of anguish and suffocation, and life may be prolonged for several hours. In a Montreal case, which I examined, the patient walked up a steep hill after the onset of the symptoms, and lived for thirteen hours. A case is on record in which the patient lived for eleven days.

New Growths and Parasites.

Tubercle and syphilis have already been considered. Primary cancer or sarcoma is extremely rare. Secondary tumors may be single or multiple, and are usually unattended with symptoms, even when the disease is most extensive. In one case I found in the wall of the right ventricle a mass which involved the anterior segment of the tricuspid valve and partly blocked the orifice. The surface was eroded and there were numerous cancerous emboli in the pulmonary artery. In another instance the heart was greatly enlarged, owing to the presence of innumerable masses of colloid cancer the size of cherries. The mediastinal sarcoma may penetrate the heart, though it is remarkable how extensive the disease of the mediastinal glands may be without involvement of the heart or vessels.

Cysts in the heart are rare. They are found in different parts, and are filled either with a brownish or a clear fluid. Blood-cysts occasionally occur.

The parasites have been discussed under the appropriate section, but it may be mentioned here that both the *cysticerus cellulosæ* and the echinococcus cysts occur occasionally in the heart.

Wounds and Foreign Bodies.

Wounds of the heart are usually fatal, although there are many instances in which recovery has taken place. Bullets have been found encysted inside the ventricle. A majority of the cases of gunshot wounds, however, are necessarily fatal. Puncture of the heart by a sharp-pointed body, such as a needle or a stiletto, does not always prove fatal. Peabody has reported a case in which a pin was found embedded in the left ventricle. Suicide has been attempted by passing a needle or pin into the heart. This is not, however, necessarily fatal. Moxon mentioned a case, at the Clinical Society of London, in which a medical student, while on a spree, passed a pin into his heart. The pericardium was opened, and the head of the pin was found outside of the right ventricle. It was grasped

Paer. Rom. 1173 II. 2 1338

Wounds of the heart may be cured & normal regress.
as stabs & bullet wounds. by progression under passing from
the pulled or resorption to a glandulose for the vascular pericardium
1) Bullet wnds. of the heart are common. In W. wars, of the Rel. W.
are more. Recov. [illegible]
found Encysted in [illegible] Stab wounds are
still more common. Suicide &c. [illegible] Chemical
student &c.

Surgical Anesthesia

The employment of anesthesia with shock in the heart. In recent
State [illegible] I was called [illegible] to suture the heart & attempt
a [illegible] [illegible] removed. Hill, of Montgomery, Ala.
[illegible] ~~heart~~ [illegible] Resolution to suture wounds
I received. (Med Rec. 12.13.90

2)

3 [illegible]

Not only has the heart been sutured, but in a case of
stab wound, Sagenstecher tied the left coronary ar-
tery, which had been divided.

and an attempt made to remove it, but it was withdrawn into the heart and, it is said, caused the patient no further trouble. Hysterical girls sometimes swallow pins and needles, which, passing through the œsophagus and stomach, are found in various parts of the body. A remarkable case is reported by Allen J. Smith of a girl from whom several dozen needles and pins were removed, chiefly from subcutaneous abscesses. Several years later she developed symptoms of chronic heart-disease. At the post mortem needles were found in the tissues of the adherent pericardium, and between thirty and forty were embedded in the thickened pleural membranes of the left side.

Puncture of the heart has been recommended as a therapeutic procedure to stimulate it to action, as in chloroform narcosis, and experimental evidence has been brought forward by B. A. Watson in favor of the operation. He advises abstraction of blood in combination with the puncture—cardiocentesis. The proceeding is not without risk. Hæmorrhage may take place from the puncture, though it is not often extensive. Sloane has recently urged its use in all cases of asphyxia and in suffocation by drowning and from coal-gas. The successful case which he reports illustrates forcibly its stimulating action.

V. NEUROSES OF THE HEART.

Palpitation.

In health we are unconscious of the action of the heart. In some people one of the first indications of debility or overwork is the consciousness of the cardiac pulsations, which may, however, be perfectly regular and orderly. This is not palpitation. The term is properly limited to irregular or forcible action of the heart perceptible to the individual.

Etiology.—The expression "perceptible to the individual" covers the essential element in palpitation of the heart. The most extreme disturbance of rhythm, a condition even of what is termed *delirium cordis*, may be unattended with subjective sensations of distress, and there may be no consciousness of disturbed action. On the other hand, there are cases in which complaint is made of the most distressing palpitation and sensations of throbbing, in which the physical examination reveals a regularly acting heart, the sensations being entirely subjective. We meet with this symptom in a large group of cases in which there is increased excitability of the nervous system. Palpitation may be a marked feature at the time of puberty, at the climacteric, and occasionally during menstruation. It is a very common symptom in hysteria and neurasthenia, particularly in the form of the latter which is associated with dyspepsia. Emotions, such as fright, are common causes of palpitation. It may occur as a sequence of the acute fevers. Females are more liable to the affection than males.

In a second group the palpitation results from the action upon the heart of certain substances, such as tobacco, coffee, tea, and alcohol. And, lastly, palpitation may be associated with organic disease of the heart, either of the myocardium or of the valves. As a rule, however, it is a

purely nervous phenomenon—seldom associated with organic disease—in which the most violent action and the most extreme irregularity may exist without that subjective element of consciousness of the disturbance which constitutes the essential feature of palpitation.

The irritable heart described by Da Costa, which was so common among the young soldiers during the civil war, is a neurosis of this kind. The chief symptoms were palpitation with great frequency of the pulse on exertion, a variable amount of cardiac pain, and dyspnœa. The factors at work in producing this condition appeared to be the mental excitement, the unwonted muscular exertion associated with the drill, and diarrhœa. The condition is not infrequent in civil life among young men, and it leads in some cases to hypertrophy of the heart.

Symptoms.—In the mildest form, such as occurs during a dyspeptic attack, there is slight fluttering of the heart and a sense of what patients sometimes call "goneness." In more severe attacks the heart beats violently, its pulsations against the chest wall are visible, the rapidity of the action is much increased, the arteries throb forcibly, and there is a sense of great distress. In some instances the heart's action is not at all quickened. The most striking cases are in neurasthenic women, in whom the mere entrance of a person into the room may cause the most violent action of the heart and throbbing of the peripheral arteries. The pulse may be rapidly increased until it reaches 150 or 160. A diffuse flushing of the skin may appear at the same time. After such attacks, there may be the passage of a large quantity of pale urine. In many cases of palpitation, particularly in young men, the condition is at once relieved by exertion. A patient with extreme irregularity of the heart may, after walking quickly 100 yards or running upstairs, return with the pulse perfectly regular. This is not infrequently seen, too, in the irregular action of the heart in mitral valve disease.

The physical examination of the heart is usually negative. The sounds, the shock of which may be very palpable, are on auscultation clear, ringing, and metallic, but not associated with murmurs. The second sound at the base may be greatly accentuated. A murmur may sometimes be heard over the pulmonary artery or even at the apex in cases of rapid action in neurasthenia or in severe anæmia. The attacks may be transient, lasting only for a few minutes, or may persist for an hour or more. In some instances any attempt at exertion renews the attack.

The *prognosis* is usually good, though it may be extremely difficult to remove the conditions underlying the palpitation.

Arrhythmia.

An intermission occurs when one or more beats of the heart are dropped. Irregularity is the condition when the beats are unequal in volume and force, or follow each other at unequal distances. Allorrhythmia is a term which is also used to express deviations from the normal heart rhythm.

The following varieties of arrhythmical action may be recognized:

(1) The paradoxical pulse of Kussmaul, in which the beats during in-

Dr. Clarence H. Vaught
SURGEON FOR
LOUISVILLE AND NASHVILLE R.R.
RICHMOND, KY.

July 25th 1900.

Dr Wilmer Osler.
Johns Hopkins Hospital
Baltimore
Md

My Dear Doctor,

I trust you will pardon me for having availed myself of your kindness in asking your opinion in the case of a little friend of mine. I have treated him for a severe attack of acute bronchitis and lung trouble, which occurred about the 1st of last January. He has literally survived death for weeks together, and has been under the care of two physicians, both of whom pronounced the case one of tuberculosis. He has had a severe hemorrhage from the lungs, and has been confined to his bed for more than a month. He is now [illegible] and I have treated him for a tubercular condition, but during the last few weeks he has had a severe cough which

spiration are more frequent but less full than during expiration. This is found in weak heart, in chronic pericarditis, and when fibrous bands encircle the root of the aorta; but it may also occur normally from the influence of the respirations upon the heart. It is sometimes to be felt in sleeping children.

(2) Intermittence, in which there is simply an intermission or dropping of a cardiac beat. The term *deficience* is more correctly applied to those instances in which the absence of the heart-sound proves that the systole is really omitted. The systole may be so weak as not to produce a pulsation, and yet at the same time a feeble first sound may be heard.

(3) The alternate heart-beat, in which strong and weak contractions alternate regularly and which is expressed in the peripheral arteries by alternate full and feeble pulse-beats.

(4) The bigeminal and trigeminal pulsations occur when two or three beats follow each other in rapid succession, each group being separated from the following by a longer interval. This is not very uncommon in mitral disease and as an effect of digitalis. In the bigeminal pulse the first beat of the pair is usually the stronger. Indeed, in the condition known as heart bigeminism the second systole is so feeble that the pulse wave does not reach the peripheral arteries and the two systoles are represented by only a single pulse-beat at the wrist.

(5) Delirium cordis, in which these various factors are combined and the heart's action is wholly irregular.

(6) Fœtal heart rhythm—embryocardia—described by Stokes, is a very common condition in which the long pause is shortened and the characters of the sounds are "almost completely identical." The resemblance to the fœtal heart-beat is very striking. In the later stages of fevers and in extreme dilatation this form of heart rhythm is very frequently heard.

(7) Gallop rhythm, in which the sounds resemble the footfall of a horse at canter, usually results from the reduplication of the sounds in a rapidly acting heart. It is expressed by the words "rat-ta-tat." Sometimes it seems as if the first sound was split; more commonly it is the second. It is most frequently heard in the failing heart of interstitial nephritis and arterio-sclerosis. Its mode of origin has been much discussed, and it is doubtful whether a satisfactory explanation has yet been given. As Graham Steell states, its presence indicates muscle weakness. It is interesting among disturbances of rhythm as the only one which we can see and feel as well as hear.

The causes of these various disturbances of rhythm are thus classified by G. Baumgarten: *

(1) Those due to central—cerebral—causes, either organic disease, as in hæmorrhage, or concussion; more commonly psychical influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of

* Transactions of the Association of American Physicians, vol. iii.

Dr. Clarence H. Vaught

SURGEON FOR

LOUISVILLE AND NASHVILLE R.R.

RICHMOND, KY.

Heart in text book.

Cons.

d. 600.

arrhythmia. Various drugs, such as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (*a*) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort and may have an important influence in producing disturbances in the rhythm; but as yet we do not know their exact significance. They may be present in cases which have not presented arrhythmia. (*b*) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter usually associated with sclerosis of the coronary arteries.

The significance of arrhythmia is not always easy to determine. Simple irregular action of the heart may persist for years. The late Chancellor Ferrier, of McGill University, a man of unusual bodily and mental vigor, who died at the age of eighty-seven, had an extremely irregular pulse for almost fifty years of his life. One or two other instances have come under my notice of persons in good health, without arterial or cardiac disease, in whom the heart's action was persistently irregular. The bigeminal and trigeminal pulsations are found more frequently in mitral than in other conditions. The delirium cordis is met with in the dilatation associated with valvular lesions, particularly toward the latter stages. Fœtal heart rhythm is rarely found apart from dilatation.

Rapid Heart—Tachycardia.

The rapid action may be perfectly natural. There are individuals whose normal heart action is at 100 or even more per minute. It may be caused by the various conditions which induce palpitation; but the two are not necessarily associated. Emotional causes, violent exercise, and fevers all produce great increase in the rapidity of the heart's action. The extremely rapid action which follows fright may persist for days, or even weeks. Traube reports an instance in which, after violent exercise, the rapidity of the heart continued. Cases are not uncommon at the menopause.

There are cases again in which the condition can hardly be termed a neurosis, since it depends upon definite changes in the pneumogastrics or in the medulla. Cases have been reported in which tumor or clot in or about the medulla or pressure upon the vagi has been associated with heart hurry. Some of the cases of frequent action of the heart in women have been thought to be due to reflex irritation from ovarian or uterine disease.

Paroxysmal tachycardia is a remarkable affection, characterized by spells of heart hurry, during which the action is greatly increased, the pulse reaching 200 and over. The cases are not common. The condition has been thoroughly studied by Nothnagel. The attack may be quite short and persist only for an hour or so. A patient at the Philadelphia Infirmary for Nervous Diseases was attacked every week or two; the pulse would rise to 220 or 230, and there were such feelings of distress and uneasiness that the patient always had to lie down. There may be, however, no subjective

University of Pennsylvania.

THE COLLEGE.

OFFICE OF THE DEAN.

WILLIAM A. LAMBERTON, DEAN.
JOSIAH H. PENNIMAN, VICE-DEAN.

PHILADELPHIA,

March 2d, 1897.

My dear Dr Cameron: Your letter of Jan 29th somehow got mislaid. I should be very glad to help you, if I can; but a lay reader, as you know, is very apt to put his foot in it from lack of certain points of knowledge, that are quite elementary to professional men.

βραδύς is first 'slow' + then is used of an interval that contains (few) units by count: βραδύς is slow, slow-moving. κρόνος is a general word for a 'slow' or 'beat' (as in music). The regular word for 'pulse' is σφυγμός. If what you want is to indicate sluggishness, low rate of movement, βραδύς is the adjective; if a small number of movements in a given interval (βραδύς: very likely they could touch the same thing. The use of κρόνος or σφυγμός seems to have this advantage over κρόνος that it indicates specifically 'heart-action'.

Yours very truly,

W. A. Lamberton,

disturbance, and in another case the patient was able to walk about during the paroxysm and had no dyspnœa. One of the most remarkable cases is reported by H. C. Wood. A physician in his eighty-seventh year had had attacks at intervals since his thirty-seventh year. The onset was abrupt and the pulse would rapidly rise to 200 a minute. For more than twenty years the taking of ice-water or strong coffee would arrest the attacks. Bouveret has analyzed a number of cases of this essential or idiopathic form; he finds that a permanent cure is rare, and that the patients suffer for ten or more years. Four instances terminated fatally from heart-failure. Martius looks upon it as a symptom of an acute dilatation of the heart, appearing paroxysmally. Wood suggests that these cardiac paroxysms are caused by discharging lesions affecting the centres of the accelerator nerves. François Franck has shown that the acceleration of the heart's action is due to the shortening of the diastole, and during the systole so little blood is expelled from the heart that the average amount in the minute is not increased. Moreover, the accelerators appear to have no trophic relation to the heart, and stimulation of them is not accompanied either by increased arterial pressure or by augmentation of the work done by the heart.

Slow Heart—Brachycardia (*Bradycardia*).

Slow action of the heart is sometimes normal and may be a family peculiarity. Napoleon is stated to have had a pulse of only 40 per minute.

In any case of slow pulse it is important first to make sure that the number of heart and arterial beats correspond. In many instances this is not the case, and with a radial pulse at 40 the cardiac pulsations may be 80, half the beats not reaching the wrist. The heart contractions, not the pulse wave, should be taken into account. A most exhaustive study of this condition has been made by Riegel, whose division is here followed:

(*a*) Physiological brachycardia. In the puerperal state the pulse may beat from 44 to 60 per minute, or may even be as low as 34. It is seen in premature labor as well as at term. The explanation of its occurrence at this period is not clear. Slowness of the pulse is associated with hunger. Brachycardia depending on individual peculiarity is extremely rare.

(*b*) Pathological brachycardia, which is met with under the following conditions: (1) In convalescence from acute fevers. This is extremely common, particularly after pneumonia, typhoid fever, acute rheumatism, and diphtheria. It is most frequently seen in young persons and in cases which have run a normal course. Traube's explanation that it is due to exhaustion is probably the correct one. (2) In diseases of the digestive system, such as chronic dyspepsia, ulcer or cancer of the stomach, and jaundice. The largest number of Riegel's cases were of this group. (3) In diseases of the respiratory system. Here it is by no means so common, but is seen not infrequently in emphysema. (4) In diseases of the circulatory system. Excluding all cases of irregularity of the heart, brachycardia is not common in diseases of the valves. It is most frequently seen in fatty and fibroid changes in the heart, but is not constant in them. (5) In diseases of the urinary organs. It occurs occasionally in nephritis and

Form 59-4000-7-23-96.

THE JOHNS HOPKINS HOSPITAL,
NORTH BROADWAY.

HENRY M. HURD, M. D.
Superintendent.

Baltimore, Jan. 29th. 189 7

My dear Professor Lamberton:-

May I ask your help in reference to the etymology of two words used in medicine?

"Brachycardia" and "Bradycardia" are used indiscriminately to signify a slow heart.

Liddell and Scott give βραχύς in reference to Space and Time as meaning "Short" of number and degree "Few" or "Little", the Latin equivalent being "Brevibus" or "Paucis".

Leverett gives the equivalent of "Paucis" ὀλιγος which is the word used all through medical nomenclature to signify few in number.

βραδύς (Liddell & Scott) is given as "Slow".

Although these terms are applied to the pulse yet the reference is primarily to the heart's action. Is it perhaps more correct to speak of a pulse with few beats to the minute as "Brachykrousis" and the heart producing such a pulse as "Bradycardia"?

Brachykrousis I have never seen used. I have added κρουσις meaning "impulse" to the limiting word βραχύς which would it seems in its meaning of "Few", have more reference to the pulse than to the heart. [redacted]

Trusting that I am not troubling you too much I remain very sincerely
Your former pupil
Charles N. Bendelari Camac

THE JOHNS HOPKINS HOSPITAL.
NORTH BROADWAY

may be a feature of uræmia. (6) From the action of toxic agents. It occurs in uræmia, poisoning by lead, alcohol, and follows the use of tobacco, coffee, and digitalis. (7) In constitutional disorders, such as anæmia, chlorosis, and diabetes. (8) In diseases of the nervous system. Apoplexy, epilepsy, the cerebral tumors, affections of the medulla, and diseases and injuries of the cervical cord may be associated with very slow pulse. In general paresis, mania, and melancholia it is not infrequent. (9) It occurs occasionally in affections of the skin and sexual organs, and in sunstroke, or in prolonged exhaustion from any cause.

The Stokes-Adams Syndrome.—*Slow Pulse with Syncopal Attacks.*—Robert Adams and Stokes described a remarkable condition in which the pulse was permanently slow in association with attacks of syncope. The patients are usually advanced in years and show an extreme grade of arteriosclerosis. The pulse-rate may be 30 or 20 to the minute, or, as in Prentice's case, as low as 12, or even 10 or 5. The cerebral symptoms are very remarkable, and Stokes suggested for them the name of false or pseudo-apoplexy. Attacks of vertigo, which may recur several times in the day, attacks of syncope, in which the patient is insensible for four or five minutes, or epileptiform attacks, as in Ogle's cases, are the most pronounced cerebral symptoms. Huchard regards the condition as the result of changes in the pneumogastric centres due to disease of the arteries of the medulla. (See Lecture IV in my monograph on Angina Pectoris and Allied States.)

Treatment of Palpitation and Arrhythmia.—An important element in many cases is to get the patient's mind quieted, and he can be assured that there is no actual danger. The mental element is oftentimes very strong. In palpitation, before using medicines, it is well to try the effect of hygienic measures. As a rule, moderate exercise may be taken with advantage. Regular hours should be kept, and at least ten hours out of the twenty-four should be spent in the recumbent posture. A tepid bath may be taken in the morning, or, if the patient is weakly and nervous, in the evening, followed by a thorough rubbing. Hot baths and the Turkish bath should be avoided. The dietetic management is most important. It is best to prohibit absolutely alcohol, tea, and coffee. The diet should be light and the patient should avoid taking large meals. Articles of food known to cause flatulency should not be used. If a smoker, the patient should give up tobacco. Sexual excitement is particularly pernicious, and the patient should be warned specially on this point. For the distressing attacks of palpitation which occur with neurasthenia, particularly in women, a rigid Weir Mitchell course is the most satisfactory. It is in these cases that we find the most distressing throbbing in the abdomen, which is apt to come on after meals, and is very much aggravated by flatulency. The cases of palpitation due to excesses or to errors in diet and dyspepsia are readily remedied by hygienic measures.

A course of iron is often useful. Strychnia is particularly valuable, and is perhaps best administered as the tincture of nux vomica in large doses. Very little good is obtained from the smaller quantities. It should be given freely, 20 minims three times a day.

If there is great rapidity of action, aconite may be tried or veratrum

Harry. by Power. p 226

case of acalgesia pedoris

Sir Robt. Darcy — when he had reached
about the middle period of life, made a
frequent complaint of a certain dull pressive
pain in the chest especially in the night
season, & that during at one time
by night & not another suffocation in his
throat &c." Died in a paroxy. Kept low
until [illegible]

226 another case. Case perhaps
acalgesia & another

viride. There are cases associated with sleeplessness and restlessness which are greatly benefited by bromide of potassium. Digitalis is very rarely indicated, but in obstinate cases it may be tried with the nux vomica.

Cases of heart hurry are often extremely obstinate, as may be judged from the case of the physician reported by H. C. Wood, in whom the condition persisted in spite of all measures for fifty years. The bromides are sometimes useful; the general condition of neurasthenia should be treated, and during the paroxysm an ice-bag may be placed upon the heart, or Leiter's coil, through which ice-water may be passed. Electricity, in the form of galvanism, is sometimes serviceable, and for its mental effect the Franklinic current. For the condition of slow pulse but little can be done. A great majority of the cases are not dangerous.

Angina Pectoris.

Stenocardia, or the breast-pang, described by Heberden, is not an independent affection, but a symptom associated with a number of morbid conditions of the heart and vessels, more particularly with sclerosis of the root of the aorta and changes in the coronary arteries. True angina, which is a rare disease, is characterized by paroxysms of agonizing pain in the region of the heart, extending into the arms and neck. In violent attacks there is a sensation of impending death.

Etiology.—It is a disease of adult life and occurs almost exclusively in men. In Huchard's statistics of 237 cases only 42 were in women. In my series of 40 cases there was only one woman. It may occur through several generations, as in the Arnold family. Gout, diabetes, and syphilis are important factors. A number of cases of angina pectoris have followed influenza. Attacks are not infrequent in certain forms of heart-disease, particularly aortic insufficiency and adherent pericardium. It is much less common in disease of the mitral valve. Almost without exception the subjects of true angina have arterio-sclerosis, either general or localized, at the root of the aorta, with changes in the coronary arteries and in the myocardium.

Phenomena of the Attack.—The exciting cause is in a majority of all cases well defined. In only rare instances do the patients have attacks when quiet. They come on during exertion most frequently, as in walking up hill or something entailing sudden muscular effort; occasionally even the effort of dressing or of stooping to lace the shoes may bring on a paroxysm. Mental emotion is a second very potent cause. John Hunter appreciated this when he said that "his life was in the hands of any rascal who chose to annoy and tease him." In his case a fatal attack occurred during a fit of anger. A third, and in many instances the most important, factor is flatulent distention of the stomach. Another common exciting cause is cold; even the chill of getting out of bed in the morning or on bathing may bring on a paroxysm.

Usually during exertion or intense mental emotion the patient is seized with an agonizing pain in the region of the heart and a sense of constriction, as if the heart had been seized in a vice. The pains radiate up the

neck and down the arm, and there may be numbness of the fingers or in the cardiac region. The face is usually pallid and may assume an ashy-gray tint, and not infrequently a profuse sweat breaks out over the surface. The paroxysm lasts from several seconds to a minute or two, during which, in severe attacks, the patient feels as if death were imminent. As pointed out by Latham, there are two elements in the paroxysm, the pain—*dolor pectoris*—and the indescribable feeling of anguish and sense of imminent dissolution—*angor animi*. There are great restlessness and anxiety, and the patient may drop dead at the height of the attack or faint and pass away in syncope. The condition of the heart during the attack is variable; the pulsations may be uniform and regular. The pulse tension, however, is usually increased, but it is surprising, even in cases of extreme severity, how slightly the character of the pulse may be altered. After the attack there may be eructations, or the passage of a large quantity of clear urine. The patient usually feels exhausted, and for a day or two may be badly shaken; in other instances in an hour or two the patient feels himself again. While dyspnœa is not a constant feature, the paroxysm is not infrequently associated with a form of asthma; there is wheezing in the bronchial tubes, which may come on very rapidly, and the patient gets short of breath. Many patients the subject of angina die suddenly without warning and not in a paroxysm. In other instances death follows in the first well-marked paroxysm, as in the case of Thomas Arnold. In a third group there are recurring attacks over long periods of years, as in John Hunter's case; while in a fourth group of cases there are rapidly recurring attacks for several days in succession, with progressive and increasing weakness of the heart.

With reference to the radiation of pain in angina, the studies of Mackenzie and of Head are of great interest. Head concludes that (1) in diseases of the heart, and more particularly in aortic disease, the pain is referred along the first, second, third, and fourth dorsal areas; (2) in angina pectoris the pain may be referred in addition along the fifth, sixth, and seventh, and even the eighth and ninth dorsal areas, and is always accompanied by pain in certain cervical areas.

Theories of Angina Pectoris.—(1) That it is a neuralgia of the cardiac nerves. In the true form the agonizing cramp-like character of the pain, the suddenness of the onset, and the associated features, are unlike any neuralgic affection. The pain, however, is undoubtedly in the cardiac plexus and radiates to adjacent nerves. It is interesting to note, in connection with the almost constant sclerosis of the coronary arteries in angina, that Thoma has found marked sclerosis of the temporal artery in migraine and Dana has met with local thickening of the arteries in some cases of neuralgia. (2) Heberden believes that it was a cramp of the heart-muscle itself. Cramp of certain muscular territories would better explain the attack. (3) That it is due to the extreme tension of the ventricular walls, in consequence of an acute dilatation associated, in the majority of cases, with affection of the coronary arteries. Traube, who supported this view, held that the agonizing pain resulted from the great stretching and tension of the nerves in the muscular substance. A modi-

fied form of this view is that there is a spasm of the coronary arteries with great increase of the intracardiac pressure.

(4) The theory of Allan Burns, revived by Potain and others, that the condition is one of transient ischæmia of the heart-muscle in consequence of disease, or spasm, of the coronary arteries. The condition known as intermittent claudication illustrates what may take place. In man (and in the horse), in consequence of thrombosis of the abdominal aorta or iliacs, transient paraplegia and spasm may follow exertion. The collateral circulation, ample when the limbs are at rest, is insufficient after the muscles are actively used, and a state of relative ischæmia is induced with loss of power, which disappears in a short time. This "intermittent claudication" theory has been applied to explain the angina paroxysm. A heart the coronary arteries of which are sclerotic or calcified, is in an analogous state, and any extra exertion is likely to be followed by a relative ischæmia and spasm. In Allan Burns's work on The Heart (1809) the theory is discussed at length, but he does not think that spasm is a necessary accompaniment of the ischæmia.

In fatal cases of angina the coronary arteries are almost invariably diseased either in their main divisions, or there is chronic endarteritis with great narrowing of the orifices at the root of the aorta. Experimentally, occlusion of the coronary arteries produces slowing of the heart's action, gradual dilatation, and death within a very few minutes. Cohnheim has shown that in the dog ligation of one of the large coronary branches produces within a minute a condition of arrhythmia, and within two minutes the heart ceases in diastole. These experiments, however, do not throw much light upon the etiology of angina pectoris. Extreme sclerosis of the coronary arteries is common, and a large majority of the cases present no symptoms of angina. Even in the cases of sudden death due to blocking of an artery, particularly the anterior branch of the coronary artery, there is usually no great pain either before or during the attack.

Diagnosis.—There are many grades of true angina. A man may have slight præcordial pain, a sense of distress and uneasiness, and radiation of the pains to the arm and neck. Such attacks following slight exertion, an indiscretion in diet, or a disturbing emotion, may alternate with attacks of much greater severity, or they may occur in connection with a pulse of increased tension and signs of general arterio-sclerosis. In the milder grades the diagnosis cannot rest upon the symptoms of the attack itself, since they may be simulated by the pseudo-angina; but the diagnosis should be based upon the examination of the heart and arteries and a careful consideration of the mode of onset and symptoms. The cases of pseudo-angina pectoris in women are, after all, the ones which call for the greatest care in the diagnosis, and attention to the points given in the table of Huchard will be of the greatest aid.

Pseudo-Angina Pectoris.—False angina may be divided into two main groups, the neurotic and the toxic. The former embraces the hysterical and neurasthenic cases, which are very common in women. Huchard has given an excellent differential table between the true and the spurious attacks.

TRUE ANGINA.	PSEUDO-ANGINA.
Most common between the ages of forty and fifty years.	At every age, even six years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely periodical or nocturnal.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with nervous symptoms.
Vaso-motor form rare. Agonizing pain and sensation of compression by a vice.	Vaso-motor form common. Pain less severe; sensation of distention.
Pain of short duration. Attitude: silence, immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions: sclerosis of coronary artery.	Neuralgia of nerves and cardio-plexus.
Prognosis grave, often fatal.	Never fatal.
Arterial medication.	Antineuralgic medication.

A form which Nothnagel has described as *vaso-motor angina* is not infrequent. The symptoms set in with coldness and numbness in the extremities, followed by great præcordial pain and feelings of faintness. Some have recognized also a reflex variety.

Toxic Angina.—This embraces cases due to the abuse of tea, coffee, and tobacco. There are three groups of cases of so-called tobacco heart: First, the irritable heart of smokers, seen particularly in young lads, in which the symptoms are palpitation, irregularity, and rapid action; secondly, heart pain of a sharp, shooting character, which may be very severe; and, thirdly, attacks of such severity that they deserve the name of angina. Huchard remarks that they are usually of the vaso-motor type, accompanied with chilling of the extremities, feeble pulse, and a tendency to syncope. This author distinguishes between functional tobacco angina, due, he thinks, to spasmodic contraction of the coronary arteries, and an organic tobacco angina due to a nicotine arterio-sclerosis of these vessels.

Prognosis.—Cardiac pain without evidence of arterio-sclerosis or valve-disease is not of much moment. True angina is almost invariably associated with marked cardio-vascular lesions, in which the prognosis is always grave. With judicious treatment the attacks, however, may be long deferred, and a few instances recover completely. The prognosis is naturally more serious with aortic insufficiency and advanced arterio-sclerosis. Patients who have had well-marked attacks may live for many years, but much depends upon the care with which they regulate their daily life.

Treatment.—Patients subject to this affection should live a quiet life, avoiding particularly excitement and sudden muscular exertion. During the attack nitrite of amyl should be inhaled, as advised by Lauder Brunton. From 2 to 5 drops may be placed upon cotton-wool in a tumbler or upon the handkerchief. This is frequently of great service in the attack, relieving the agonizing pain and distress. Subjects of the dis-

In the new-born particularly if premature, there is a possibility that a systolic basic murmur, even of great intensity, may originate in the ductus Botalli before its closure. (Cacherich-Jacobi Festschrift).

ease should carry the *perles* of the nitrite of amyl with them, and use them on the first indication of an attack. In some instances the nitrite of amyl is quite powerless, though given freely. If within a minute or two relief is not obtained in this way, chloroform should at once be given. A few inhalations act promptly and give great relief. Should the pains continue, a hypodermic of morphia may be administered. In severe and repeated paroxysms a patient may display remarkable resistance to the action of this drug.

In the intervals, nitroglycerin may be given in full doses, as recommended by Murrell, or the nitrite of sodium (Matthew Hay). The nitroglycerin should be used for a long time and in increasing doses, beginning with 1 minim three times a day of the 1-per-cent solution, and increasing the dose 1 minim every five or six days until the patient complains of flushing or headache. The fluid extract of English hawthorn—crategus oxyacantha—has been strongly recommended by Jennings, Clements, and others.

Huchard recommends the iodides, believing that their prolonged use influences the arterio-sclerosis. Twenty grains three times a day may be given for several years, omitting the medicine for about ten days in each month. In some instances this treatment is certainly beneficial. Two men, both with arterio-sclerosis, ringing, accentuated aortic sound, and attacks of true angina, have under its use remained practically free from attacks—one case for nearly three, and the other for fully eight years. This treatment is, however, not always satisfactory, and I have had several cases in which the condition has not been at all relieved by it.

For the pseudo-angina, the treatment must be directed to the general nervous condition. Electricity is sometimes very beneficial, particularly the Franklinic form.

VI. CONGENITAL AFFECTIONS OF THE HEART.

These have only a limited clinical interest, as in a large proportion of the cases the anomaly is not compatible with life, and in others nothing can be done to remedy the defect or even to relieve the symptoms.

The congenital affections result from interruption of the normal course of development or from inflammatory processes—endocarditis; sometimes from a combination of both.

(*a*) Of *general anomalies* of development the following conditions may be mentioned: *Acardia*, absence of the heart, which has been met with in the monstrosity known by the same name; *double heart*, which has occasionally been found in extreme grades of fœtal deformity; *dextrocardia*, in which the heart is on the right side, either alone or as part of a general transposition of the viscera; *ectopia cordis*, a condition associated with fission of the chest wall and of the abdomen. The heart may be situated in the cervical, pectoral, or abdominal regions. Except in the abdominal variety the condition is very rarely compatible with extra-uterine life. Occasionally, as in a case reported by Holt, the child lives for some months,

and the heart may be seen and felt beating beneath the skin in the epigastric region. This infant was five months old at the date of examination.

(*b*) **Anomalies of the Cardiac Septa.**—The septa of both auricles and ventricles may be defective, in which case the heart consists of but two chambers, the *cor biloculare* or reptilian heart. In the septum of the auricles there is a very common defect, owing to the fact that the membrane closing the foramen ovale has failed at one point to become attached to the ring, and leaves a valvular slit which may be large enough to admit the handle of a scalpel. Neither this nor the small cribriform perforations of the membrane are of any significance.

The foramen ovale may be patent without a trace of membrane closing it. In some instances this exists with other serious defects, such as stenosis of the pulmonary artery, or imperfection of the ventricular septum. In others the patent foramen ovale is the only anomaly, and in many instances it does not appear to have caused any embarrassment, as the condition has been found in persons who have died of various affections. The ventricular septum may be absent, the condition known as trilocular heart. Much more frequently there is a small defect in the upper portion of the septum, either in the situation of the membranous portion known as the "undefended space" or in the region situated just anterior to this. The anomaly is very frequently associated with narrowing of the pulmonary orifice or of the conus arteriosus of the right ventricle.

(*c*) **Anomalies and Lesions of the Valves.**—Numerical anomalies of the valves are not uncommon. The semilunar segments at the arterial orifices are not infrequently increased or diminished in number. Supernumerary segments are more frequent in the pulmonary artery than in the aorta. Four, or sometimes five, valves have been found. The segments may be of equal size, but, as a rule, the supernumerary valve is small.

Instead of three there may be only two semilunar valves, or, as it is termed, the *bicuspid condition*. In my experience, this is most frequent in the aortic valve. Of 21 instances only 2 occurred at the pulmonary orifice. Two of the valves have united, and from the ventricular face show either no trace of division or else a slight depression indicating where the union had occurred. From the aortic side there is usually to be seen some trace of division into two sinuses of Valsalva. There has been a discussion as to the origin of this condition, whether it is really an anomaly or whether it is not due to endocarditis, fœtal or post-natal. The combined segment is usually thickened, but the fact that this anomaly is met with in the fœtus without a trace of sclerosis or endocarditis shows that it may, in some cases at least, result from a developmental error.

Clinically this is a very important congenital defect, owing to the liability of the combined valve to sclerotic changes. Except two fœtal specimens all of my cases showed thickening and deformity, and in 15 of those which I have reported death resulted directly or indirectly from the lesion.

The little fenestrations at the margins of the sigmoid valves have no significance; they occur in a considerable proportion of all bodies.

and the heart may be seen and felt beating beneath the skin in the epigastric region. This infant was five months old at the date of examination.

(*b*) **Anomalies of the Cardiac Septa.** The septa of the auricles and

A Case of Persistent Foramen Ovale with Auscultatory Signs.

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REPRINTED FROM THE OCTOBER NUMBER
OF COLORADO MEDICAL JOURNAL.
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A Case of Persistent Foramen Ovale with Auscultatory Signs.

By HENRY SEWALL, PH. D., M. D.

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The foramen ovale is an opening in the auricular septum normally present during fœtal life. It establishes a direct communication between the two auricles and is designed to allow the current of oxygenated blood from the inferior vena cava to be diverted from a useless course through the right ventricle and lungs. It is commonly supposed that the passage of blood through the foramen ovale ceases at birth; for with the first inspiration the current of blood entering the lungs and returning to the left auricle is greatly augmented

the two auricles, even a patent opening may exist in the auricular septum without direct passage of blood from one cavity to the other or functional disturbance of the circulation. It seems reasonable to hope that circulatory failure resulting from this anatomical defect might be compensated by treatment directed toward the restoration of equilibrium of auricular pressures. The occurrence of cyanosis in a patient suffering from uncomplicated, persistent foramen ovale is prima facie evidence that venous blood is finding its way directly from the right to the left aur

and the heart may be seen and felt beating beneath the skin in the epigastric region. This infant was five months old at the date of examination.

(*b*) **Anomalies of the Cardiac Septa.**—The septa of both auricl

of persistent foramen ovale was diagnosed during life and the opinion verified by a post mortem observation.

CASE.—A female waif, apparently less than a week old, was brought to the County hospital July 17th. The infant was emaciated and wizened in appearance and its skin was slightly bluish or slate colored. The patient fed well from the bottle, but suffered later from diarrhoea, passing from eight to ten greenish stools in twenty-four hours. The diarrhoea greatly improved and the intestinal condition became nearly normal by July 31st. A rather hurried physical examination conducted soon after admission of the infant to the hospital disclosed nothing abnormal in the heart. On August 2nd, though the diarrhoea had ceased, the cyanosis of the patient was somewhat more marked than hitherto and led to a detailed examination of the heart, with the following results:

Connected with the first sound of the heart was a short, faint but distinct murmur. The unusual area of distribution and time relations of this murmur at once fixed the attention and led to the suspicion that it had its or

acter. That is to say, in contra-distinction to systolic murmurs. Its ending was abrupt, not gradual, its accent was acute, not grave. The ear can distinguish sound characters such as these when, with rapid heartbeat, the time relations of a brief murmur cannot be determined. When the murmur was heard on August 2nd it was intended to carry out more detailed observations on the next day, but the infant died during the night, probably from asthenia. The autopsy was performed by Dr. E. R. Axtell about twenty hours after death. The body was that of a female infant, considerably emaciated, about sixteen inches long, On section, all the organs appeared to be normal with the exception of the heart, whose only defect consisted in a persistent foramen ovale. The foramen consisted of an oval slit measuring about one-tenth of an inch in its long, vertical diameter, situated about the

center of the auricular septum. In the right auricle the opening was hid from view by a fold of the septum di rected backwards. In the left auricle the opening was less completely hid by muscular fibres including its posterior edge. The canal was directed from the right forwards to the left. The or

a useless course through the right ventricle and lungs. It is commonly supposed that the passage of blood through the foramen ovale ceases at birth; for with the first inspiration the current of blood entering the lungs and returning to the left auricle is greatly augmented and the pressure within the two auricles probably becomes approximately the same. Under such mechanical conditions there would be no force to maintain a current through the opening. The permanent closure of the foramen ovale proceeds slowly and is usually accomplished at some period in the first year of independent life.[1] When the foramen ovale persists unduly after birth it commonly remains not as a free opening but as an oblique slit in the auricular septum, and such a condition might obviously exist without disturbance of function. Indeed, Klob and Wallman[2] found such an opening in no less than 44 per cent. of all autopsies on adults in which the point was investigated. As already indicated, when there is equilibrium of pressures within

ment directed toward the restoration of equilibrium of auricular pressures. The occurrence of cyanosis in a patient suffering from uncomplicated, persistent foramen ovale is prima facie evidence that venous blood is finding its way directly from the right to the left auricle.

Even the existence of such a cross-current does not necessarily involve the production of recognizable murmurs. No audible murmur attends the passage of blood through the foramen ovale in fœtal life, and sounds due to this cause have been but seldom heard after birth. According to Hochsinger[3], Rauchmann found that in the rare cases in which murmurs were heard in connection with uncomplicated defects in the auricular septum, the murmurs were heard most plainly in an area including the third right costal cartilage and the base of the heart. This distribution of the abnormal sound agrees well with that found in the case here reported, in which the condition

the heart. On August 2nd, though the diarrhœa had ceased, the cyanosis of the patient was somewhat more marked than hitherto and led to a detailed examination of the heart, with the following results:

Connected with the first sound of the heart was a short, faint but distinct murmur. The unusual area of distribution and time relations of this murmur at once fixed the attention and led to the suspicion that it had its origin in a persistent foramen ovale. The murmur was heard most distinctly in a segment which included the base line of the heart and curved over to the right border of the sternum. In other words, the murmur had its greatest intensity in the area of the chest wall most closely approximating the auricles. Again, the murmur appeared to be pre-systolic both in time and char-

whose only defect consisted in a persistent foramen ovale. The foramen consisted of an oval slit measuring about one-tenth of an inch in its long, vertical diameter, situated about the center of the auricular septum. In the right auricle the opening was hid from view by a fold of the septum directed backwards. In the left auricle the opening was less completely hid by muscular fibres including its posterior edge. The canal was directed from the right forwards to the left. The orifices of the pulmonary artery and of the venæ cavæ appeared to be normal. The ductus arteriosus was closed, except for a pit about one-twentieth inch in depth communicating with the aorta.

1. Quains Anatomy, 10th Ed., Vol. 1, Pt. 1, p. 157.
2. Quoted by Hochsinger, "Auscultation des Kindlichen Herens," 1890, S. 157.
3. Hochsinger Op. Cit.

Anomalies of the auriculo-ventricular valves are not often met with.

Fœtal endocarditis may occur either at the arterial or auriculo-ventricular orifices. It is nearly always of the chronic or sclerotic variety. Very rarely indeed is it of the warty or verrucose form. There are little nodular bodies, sometimes six or eight in number, on the mitral and tricuspid segments—the nodules of Albini—which represent the remains of fœtal structures, and must not be mistaken for endocardial outgrowths. The little rounded, bead-like hæmorrhages of a deep purple color, which are very common on the heart valves of children, are also not to be mistaken for the products of endocarditis. In fœtal endocarditis the segments are usually thickened at the edges, shrunken, and smooth. In the mitral and tricuspid valves the cusps are found united and the chordæ tendineæ are thickened and shortened. In the semilunar valves all trace of the segments has disappeared, leaving a stiff membranous diaphragm perforated by an oval or rounded orifice. It is sometimes very difficult to say whether this condition has resulted from fœtal endocarditis or whether it is an error in development. In very many instances the processes are combined; an anomalous valve becomes the seat of chronic sclerotic changes, and, according to Rauchfuss, endocarditis is more common on the right side of the heart only because the valves are here most often the seat of developmental errors.

Lesions at the Pulmonary Orifice.—*Stenosis* of this orifice is one of the commonest and most important of congenital heart affections. A slow endocarditis causes gradual union of the segments and narrowing of the orifice to such a degree that it only admits the smallest-sized probe. In some of the cases the smooth membranous condition of the combined segments is such that it would appear to be the result of faulty development. In some instances vegetations develop. The condition is compatible with life for many years, and in a considerable proportion of the cases of heart-disease above the tenth year this lesion is present. With it there may be defect of the ventricular septum. Pulmonary tuberculosis is a very common cause of death. Obliteration or *atresia* of the pulmonary orifice is less frequent but a more serious condition than stenosis. It is associated with defect of the ventricular septum or patency of the foramen ovale and persistence of the ductus arteriosus with hypertrophy of the right heart. *Stenosis of the conus arteriosus* of the right ventricle exists in a considerable proportion of the cases of obstruction at the pulmonary orifice. At the outset a developmental error, it may be combined with sclerotic changes. The ventricular septum is imperfect, the foramen ovale is usually open, and the ductus arteriosus patent. These three lesions at the pulmonary orifice constitute the most important group of all congenital cardiac affections. Of 181 instances of various congenital anomalies collected by Peacock 119 cases came under this category, and, according to this author, in 86 per cent of the patients living beyond the twelfth year the lesion is at this orifice.

Congenital lesions of the aortic orifice are not very frequent. Rauchfuss has collected 24 cases of stenosis and atresia; stenosis of the left conus arteriosus may also occur, a condition which is not incompatible with pro-

longed life. Ten of the 16 cases tabulated by Dilg were over thirty years of age.

Transposition of the large arterial trunks is a not uncommon anomaly. There may be neither hypertrophy, cyanosis, nor heart murmur.

Symptoms of Congenital Heart-disease.—Cyanosis occurs in over 90 per cent of the cases, and forms so distinctive a feature that the terms "blue disease" and "morbus cæruleus" are practically synonyms for congenital heart-disease. The lividity in a majority of cases appears early, within the first week of life, and may be general or confined to the lips, nose, and ears, and to the fingers and toes. In some instances there is in addition a general dusky suffusion, and in the most extreme grades the skin is almost purple. It may vary a good deal and may only be intense on exertion. The external temperature is low. Dyspnœa on exertion and cough are common symptoms. A great increase in the number of the red corpuscles has been noted by Gibson and by Vaquez. In a case of Gibson's there were above eight millions of red blood-corpuscles to the cubic millimetre. The children rarely thrive, and often display a lethargy of both mind and body. The fingers and toes are clubbed to a degree rarely met with in any other affection. The cause of the cyanosis has been much discussed. Morgagni referred it to the general congestion of the venous system due to obstruction, and this view was supported in a paper, one of the ablest that has been written on the subject, by Moreton Stillé. Morrison's recent analysis of 75 cases of congenital heart-disease shows that closure of the pulmonary orifice and patency of the foramen ovale and the ventricular septum are the lesions most frequently associated with cyanosis, and he concludes that the deficient aëration of the blood owing to diminished lung function is the most important factor. Another view, ~~advocated by~~ William Hunter, was that the discoloration was due to the admixture in the heart of venous and arterial blood; but lesions may exist which permit of very free mixture without producing cyanosis. The question of the cause of cyanosis really cannot be considered as settled. Variot has recently made the suggestion that the cause is not entirely cardiac, but is associated with disturbance throughout the whole circulatory system, and particularly a vaso-motor paresis and malaëration of the red blood-corpuscles.

Diagnosis.—In the case of children, cyanosis, with or without enlargement of the heart, and the existence of a murmur are sufficient, as a rule, to determine the presence of a congenital heart-lesion. The cyanosis gives us no clew to the precise nature of the trouble, as it is a symptom common to many lesions and it may be absent in certain conditions. The murmur is usually systolic in character. It is, however, not always present, and there are instances on record of complicated congenital lesions in which the examination showed normal heart-sounds. In two or three instances fœtal endocarditis has been diagnosed *in gravida* by the presence of a rough systolic murmur, and the condition has been corroborated subsequent to the birth of the child. Hypertrophy is present in a majority of the cases of congenital defect. The fatal event may be caused by abscess of the brain. It is impossible in a work of this sort to enter upon elabo-

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DR. GEORGE DOCK,
14 CORNWELL PLACE,
ANN ARBOR, MICH.

Jan. 19, 1901.

Dear Dr. Osler:-

I was delighted to see in yours of the XII. 26th that you have not forgotten your promised visit to Ann Arbor, and hope you will be able to make it soon. Failing an earlier day can you not stop here before or after the St. Paul meeting?

I am glad to hear you are getting out another edition, though the '98 is still ahead of the numerous "others" that came out since then. I look in vain for things to correct, though I would like to ask whether you do not think the murmur of aortic incompetency should be described in the first place as heard down the left side of the sternum? That being more frequent here, as I see it, often gives students trouble in interpreting.

I have just recalled that in a former letter you spoke of the proof

I have just recalled that in a former letter you spoke of the great cancer book reaching me in a few days. It has not come yet, and I have thought you might think me negligent in acknowleaging.

We are all glad to know Abel was not more seriously hurt, and I hope he is quite well.

I am hard at work directing the fitting up of a new laboratory; not for students, except a few, but for the routine study of cases. Though very plain, I think I shall find it well adapted for the purpose.

With best wishes from Mrs. Dock, I am

Yours sincerely,

George Dock

rate details in differential diagnosis between the various congenital heart-lesions. I here abstract the conclusions of Hochsinger:

"(1) In childhood, loud, rough, musical heart-murmurs, with normal or only slight increase in the heart-dulness, occur only in congenital heart-disease. The acquired endocardial defects with loud heart-murmurs in young children are almost always associated with great increase in the heart-dulness. In the transposition of the large arterial trunks there may be no cyanosis, no heart-murmur, and an absence of hypertrophy.

"(2) In young children heart-murmurs with great increase in the cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(4) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and cannot be explained by the assumption of an endocarditis of the aortic valve."

Treatment.—The child should be warmly clad and guarded from all circumstances liable to excite bronchitis. In the attacks of urgent dyspnœa with lividity, blood should be freely let. Saline cathartics are also useful. Digitalis must be used with care; it is sometimes beneficial in the later stages. When the compensation fails, the indications for treatment are those of valvular disease in adults.

III. DISEASES OF THE ARTERIES.

I. DEGENERATIONS.

Fatty degeneration of the intima is extremely common, and is seen in the form of yellowish-white spots in the aorta and larger vessels. *Calcification* of the arterial wall follows fatty degeneration and sclerosis, and is associated with atheromatous changes. It occurs in the intima and the media. In the latter it produces what is sometimes known as annular calcification, which occurs particularly in the middle coat of medium-sized vessels and may convert them into firm tubes.

Hyaline degeneration may attack either the larger or the smaller vessels. In the former the intima is converted into a smooth, homogeneous substance; this is commonly an initial stage of arterio-sclerosis; here it is a transformation of the endothelial lining. Of the smaller arteries and capillaries hyaline metamorphosis is oftenest seen in the glomeruli of the kidneys. It is not to be confounded with the amyloid change which is prone to occur in the same situation. The condition is variously regarded as due to coagulation of an albuminous fluid and hyaline metamorphosis of leucocytes or of fibrin. This substance reacts like the last with Weigert's fibrin stain.

II. ARTERIO-SCLEROSIS (*Arterio-capillary Fibrosis*).

The conception of arterio-sclerosis as an independent affection—a general disease of the vascular system—is due to Gull and Sutton.

Definition.—A condition of thickening, diffuse or circumscribed, beginning in the intima, consequent upon primary changes in the media and adventitia, but which later involves the other coats. The process leads, in the larger arteries, to what is known as atheroma and to endarteritis deformans.

Etiology.—(1) As an involution process arterio-sclerosis is an accompaniment of old age, and is the expression of the natural wear and tear to which the tubes are subjected. Longevity is a vascular question, which has been well expressed in the axiom that "a man is only as old as his arteries." To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arterio-sclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondly upon the amount of wear and tear to which he has subjected it. That the former plays the most important *rôle* is shown in the cases in which arterio-sclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Thus, for instance, a man of twenty-eight or twenty-nine may have the arteries of a man of sixty, and a man of forty may present vessels as much degenerated as they should be at eighty. Entire families sometimes show this tendency to early arterio-sclerosis—a tendency

which cannot be explained in any other way than that in the make-up of the machine bad material was used for the tubing.

More commonly the arterio-sclerosis results from the bad use of good vessels, and among the circumstances which tend to produce this condition are the following:

(2) *Chronic Intoxications.*—Alcohol, lead, gout, and syphilis play an important *rôle* in the causation of arterio-sclerosis, although the precise mode of their action is not yet very clear. They may act, as Traube suggests, by increasing the peripheral resistance in the smaller vessels and in this way raising the blood tension, or possibly, as Bright taught, they alter the quality of the blood and render more difficult its passage through the capillaries.

The poison of syphilis and of gout may act directly on the arteries, producing degenerative changes in the media and adventitia.

(3) *Overeating.*—Many authors attribute an important part of the etiology of arterio-sclerosis to the overfilling of the blood-vessels which occurs when unnecessarily large quantities of food and drink are taken. Particularly is this the case in stout persons who take very little exercise.

(4) *Overwork of the muscles,* which acts by increasing the peripheral resistance and by raising the blood-pressure.

(5) *Renal Disease.*—The relation between the arterial and kidney lesions has been much discussed, some regarding the arterial degeneration as secondary, others as primary. There are certainly two groups of cases, one in which the arterio-sclerosis is the first change, and the other in which it appears to be secondary to a primary affection of the kidneys. The former occurs, I believe, with much greater frequency than has been supposed.

Morbid Anatomy.—Thoma divides the cases into *primary* arterio-sclerosis, in which there are local changes in the arteries leading to dilatation and a compensatory increase of the connective tissue of the intima; *secondary* arterio-sclerosis, due to changes in the arteries which follow increased resistance to the blood-flow in the peripheral vessels. This increased tension leads to dilatation and to slowing of the blood-stream and a secondary compensatory development of the intima.

In a study of 41 autopsies upon arterio-sclerotic cases from my wards, Councilman follows the useful division into nodular, senile, and diffuse forms.

(*a*) *Nodular Form.*—In the circumscribed or nodular variety the macroscopic changes are very characteristic. The aorta presents, in the early stages, from the ring to bifurcation, numerous flat projections, yellowish or yellowish-white in color, hemispherical in outline, and situated particularly about the orifices of the branches. In the early stage these patches are scattered and do not involve the entire intima. In more advanced grades the patches undergo atheromatous changes. The material constituting the button undergoes softening and breaks up into granular material, consisting of molecular *débris*—the so-called atheromatous abscess.

In the circumscribed or nodular arterio-sclerosis the primary alteration consists in a degeneration or a local infiltration in the media and adventitia, chiefly about the vasa vasorum. The affection is really a mesarteritis

and a periarteritis. These changes lead to the weakening of the wall in the affected area, at which spot the proliferative changes commence in the intima, particularly in the subendothelial structures, with gradual thickening and the formation of an atheromatous button or a patch of nodular arterio-sclerosis. The researches of Thoma have shown that this is really a compensatory process, and that before its degeneration the nodular button, which post mortem projects beyond the lumen, during life fills up and obliterates what would otherwise be a depression of the wall in consequence of the weakening of the media. A similar process goes on in the smaller vessels, and in any one of the smaller branches it can be readily seen on section that each patch of endarteritis corresponds to a defect in the media and often to changes in the adventitia. The condition is one which may lead to rapid dilatation or to the production of an aneurism, particularly in the early stage, before the weakened spot is thickened and strengthened by the intimal changes.

(*b*) *Senile Arterio-sclerosis.*—The larger arteries are dilated and tortuous, the walls thin but stiff, and often converted into rigid tubes. The subendothelial tissue undergoes degeneration and in spots breaks down, forming the so-called atheromatous abscess, the contents of which consist of a molecular *débris*. They may open into the lumen, when they are known as atheromatous ulcers. The greater portion of the intima may be occupied by rough calcareous plates, with here and there fissures and losses of substance, upon which not infrequently white thrombi are deposited. Microscopically there is extreme degeneration of the coats, particularly of the media. Senile atrophy of the liver and kidneys usually accompanies these changes. Senile changes are common in other organs. The heart may be small and is not necessarily hypertrophied. In 7 of 14 cases of Councilman's series there was no enlargement. Brown atrophy is common.

(*c*) *Diffuse Arterio-sclerosis.*—The process is widespread throughout the aorta and its branches, in the former usually, but not necessarily, associated with the nodular form. The subjects of this variety are usually middle-aged men, but it may occur early. Of the 27 in Councilman's series belonging to this group the majority were between the ages of forty and fifty-five. The youngest was a negro of twenty-three and the oldest a man of sixty. The affection is very prevalent among negroes; less than 50 per cent were in whites, whereas the ratio of colored to white patients in the wards is one to seven. The affection is met with in strongly built, muscular men and, as Councilman remarks, they rarely present on the autopsy table signs of general anasarca or, if œdema exists, it has come on during the last few days of life. The aorta and its branches are more or less dilated, the branches sometimes more than the trunk. The intima may be smooth and show very slight changes to the naked eye; more commonly there are scattered elevated areas of an opaque white color, some of which may have undergone atheromatous changes as in the senile form.

Microscopically in the several forms the *media* shows necrotic and hyaline changes, involving in the larger arteries both muscular and elastic elements, and the *intima* presents a great increase in the subendothelial con-

nective tissue, which is particularly marked opposite areas of advanced degeneration in the media. The small arteries—those in the kidneys, for example—show "a thickening of the wall, due to the formation of a homogeneous hyaline tissue within the muscular coat. This tissue contains but few cells, is faintly striated, and stains a light brown in the osmic acid used in the hardening solution. In many of the smallest vessels nothing can be seen of the elastic lamina, in others only fragments can be made out, in others it is preserved. . . . The muscular fibres of the media show marked atrophic changes. Fatty degeneration of the cells can be made out both in fresh sections and after hardening in Flemming's solution. The nuclei are thin and atrophic and vacuoles are sometimes seen in them. In some arteries the muscle-fibres have almost disappeared and the media is changed into a homogeneous tissue, similar to that in the thickened intima" (Councilman). The degeneration of the media is most marked in the smaller arteries. The capillaries are thickened, particularly those of the glomeruli of the kidneys, which are often obliterated and involved in extensive hyaline degeneration.

It is in this group of cases that the heart shows the most important changes. The average weight in the cases referred to was over 450 grammes, and there were two cases in which without valvular disease the weight was over 800 grammes. Fibrous myocarditis is often present, particularly when the coronary arteries are involved. The semilunar valves are sometimes opaque and sclerotic, and may be incompetent. The kidneys may show extensive sclerosis, but in many cases the changes are so slight that macroscopically they might be overlooked. They may be increased in size. The capsule is usually adherent, the surface a little rough, and very often presents atrophic areas at a lower level, of a deep-red color. Increased consistence is always present.

Sclerosis of the pulmonary artery is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral valve disease and in emphysema. Sometimes the sclerosis reaches a high grade and is accompanied with aneurismal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve. In a remarkable case of a young man of twenty-four, reported by Romberg from Curschmann's clinic, the pulmonary arteries were involved in most extensive arterio-sclerosis; the main branches were dilated, and the smaller branches were the seat of the most extreme sclerotic changes. On the other hand, the aorta and its branches were normal. The heart was greatly hypertrophied, and the clinical symptoms were those of a congenital heart affection. In many cases of arterio-sclerosis the condition is not confined to the arteries, but extends not only to the capillaries but also to the veins, and may properly be termed an *angio-sclerosis*.

Sclerosis of the veins—*phlebo-sclerosis*—is not at all an uncommon accompaniment of arterio-sclerosis, and is a condition to which of late a good deal of attention has been paid. It is seen in conditions of heightened blood-pressure, as in the portal system in cirrhosis of the liver and in the pulmonary veins in mitral stenosis. The affected vessels are usually dilated, and the intima shows, as in the arteries, a compensatory thickening, which

is particularly marked in those regions in which the media is thinned. The new-formed tissue in the endophlebitis may undergo hyaline degeneration, and is sometimes extensively calcified. In a case of fibroid obliteration of the portal vein of long standing, I found the intima of the greatly dilated gastric, splenic, and mesenteric veins extensively calcified. Without existing arterio-sclerosis the peripheral veins may be sclerotic, usually in conditions of debility, but occasionally in young persons.

Symptoms.—*Increased Tension.*—The pressure with which the blood flows in the arteries depends upon the degree of peripheral resistance and the force of the ventricular contraction. A high-tension pulse may exist with very little arterio-sclerosis; but, as a rule, when the condition has been persistent, the sclerosis and high tension are found together. The pulse wave is slow in its ascent, enduring, subsides slowly, and in the intervals between the beats the vessel remains full and firm. It may be very difficult to obliterate the pulse, and the firmest pressure on the radial or the temporal artery may not be sufficient to annihilate the pulse wave beyond the point of pressure. This is not always a sign of high tension. The anastomotic or recurrent pulse may be felt even when the tension is low, as in the early stage of typhoid fever. Pressure on the ulnar artery at once obliterates it.* The sphygmographic tracing shows a sloping, short up-stroke, no percussion wave, and a slow, gradual descent, in which the dicrotic wave is very slightly marked. It may be difficult to estimate how much of the hardness and firmness is due to the tension of the blood within the vessel, and how much to the thickening of the wall. But if, for example, when the radial is compressed with the index-finger the artery can be felt beyond the point of compression, its walls are sclerosed.

Hypertrophy of the Heart.—In consequence of the peripheral resistance and increased work the left ventricle increases in size, and some of the purest examples of simple hypertrophy occur in this condition. The chamber may be little, if at all, dilated. The apex beat is dislocated in advanced cases an inch or more beyond the nipple line. The impulse is heaving and forcible. The aortic second sound is clear, ringing, and accentuated.

The combination of increased arterial tension, a palpable thickening of the arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are signs pathognomonic of arterio-sclerosis. From this period of establishment the course of the disease may be very varied. For years the patient may have good health, and be in a condition analogous to that of a person with a well-compensated valvular lesion. There may be no renal symptoms, or there may be the passage of a larger amount of urine than normal, with transient albuminuria, and now and then hyaline tube-casts. The subsequent history is extraordinarily diverse, depending upon the vascular territory in which the sclerosis is most advanced, or upon the accidents which are so liable to happen, and the symptoms may be cardiac, cerebral, renal, etc.

(1) *Cardiac.*—The involvement of the coronary arteries may lead to the various symptoms already referred to under that section—thrombosis

* The student is referred to Ewart On the Pulse, and to his larger Heart Studies.

with sudden death, fibroid degeneration of the heart, aneurism of the heart, rupture, and angina pectoris. Angina pectoris is not uncommon, and in the true variety is almost always associated with arterio-sclerosis. A second important group of cardiac symptoms results from the dilatation which ultimately may follow the hypertrophy. The patient then presents all the symptoms of cardiac insufficiency—dyspnœa, scanty urine, and very often serous effusions. If the case has come under observation for the first time the clinical picture is that of chronic valvular disease, and the existence of a loud blowing murmur at the apex may throw the practitioner off his guard. Many cases terminate in this way.

(2) The *cerebral* symptoms of arterio-sclerosis are varied and important, and embrace those of many degenerative processes, acute and chronic (which follow sclerosis of the smaller branches), and cerebral hæmorrhage.

Transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. Recovery may be perfect. It is difficult to say upon what these attacks depend. Spasm of the arteries has been suggested, but the condition of the smallest arteries is not very favorable to this view. Peabody has recently called attention to these cases, which are more common than is indicated in the literature. Vertigo occurs frequently, and may be either simple, or is associated with slow pulse and syncopal or epileptiform attacks (Grasset, Church).

(3) *Renal* symptoms supervene in a large number of the cases. A sclerosis, patchy or diffuse, is present in a majority of the cases at the time of autopsy, and the condition is practically that of contracted kidney. It is seen in a typical manner in the senile form, and not infrequently develops early in life as a direct sequence of the diffuse variety. It is often difficult to decide clinically (and the question is one upon which good observers might not agree in a given case) whether the arterial or the renal disease has been primary.

(4) Among other events in arterio-sclerosis may be mentioned gangrene of the extremities, due either directly to endarteritis or to the dislodgment of thrombi. Respiratory symptoms are not uncommon, particularly bronchitis and the symptoms associated with emphysema.

Treatment.—In the late stages the conditions must be treated as they arise in connection with the various viscera. In the early stages, before any local symptoms are manifest, the patient should be enjoined to live a quiet, well-regulated life, avoiding excesses in food and drink. It is usually best to explain frankly the condition of affairs, and so gain his intelligent co-operation. Special attention should be paid to the state of the bowels and urine, and the secretion of the skin should be kept active by daily baths. Alcohol in all forms should be prohibited, and the food should be restricted to plain, wholesome articles. The use of mineral waters or a residence every year at one of the mineral springs is usually serviceable. If there has been a syphilitic history an occasional course of iodide of potassium is indicated, and whenever the pulse tension is high nitroglycerin may be used.

In cases which come under observation for the first time with dyspnœa, slight lividity, and signs of cardiac insufficiency, venesection is indicated.

In some instances, with very high tension, striking relief is afforded by the abstraction of 20 ounces of blood.

III. ANEURISM.

The following forms of aneurism are usually recognized:

(*a*) The *true*, in which the sac is formed of one or more of the arterial coats. This may be fusiform, cylindrical, or cirsoid (in which the dilatation is in an artery and its branches), or it may be circumscribed or sacculated. Aneurisms are usually fusiform, resulting from uniform dilatation of the vessel, or saccular.

(*b*) The *false* aneurism, in which there is rupture of all the coats, and the blood is free (or circumscribed) in the tissues.

(*c*) The *dissecting* aneurism, which results from injury or laceration of the internal coat. The blood dissects betwen the layers; hence the name, dissecting aneurism. This occurs usually in the aorta, persisting for years.

(*d*) *Arterio-venous* aneurism results when a communication is established between an artery and a vein. A sac may intervene, in which case we have what is called a varicose aneurism; but in many cases the communication is direct and the chief change is in the vein, which is dilated, tortuous, and pulsating, the condition being termed an aneurismal varix.

Etiology and Pathology.—Aneurisms arise: (*a*) By the gradual diffuse distention of the arterial coats, which have been weakened by arterio-sclerosis, particularly in its early stages, before compensatory endarteritis develops. The arch of the aorta is often dilated in this way so as to form an irregular aneurism.

(*b*) In consequence of circumscribed loss of resisting power in the media and adventitia, and often from a laceration of the media. This is the most common cause of sacculated aneurism. The laceration is frequently found in the ascending portion of the arch and occurs early in the process of arterio-sclerosis, before the compensatory thickening has taken place. Occasionally one meets with remarkable specimens illustrating the important part played by this process. The intima may also be torn. In a case of Daland's there was just above the aortic valves an old transverse tear of the intima, extending almost the entire circumference of the vessel. Sclerosis of the media and adventitia had taken place and the process was evidently of some standing. An inch or more above it was a fresh transverse rent which had produced a dissecting aneurism. These arterio-sclerotic aneurisms, as they are called, are found also in the smaller vessels.

(*c*) *Embolic Aneurism.*—When an embolus has lodged in a vessel and permanently plugged it, aneurismal dilatation may follow on the proximal side. The embolus itself may, if a calcified fragment from a valve, lacerate the wall, or if infected may produce inflammation and softening.

(*d*) *Mycotic Aneurism.*—The importance of this form has been specially considered by Eppinger in his exhaustive monograph. The occurrence of multiple aneurisms in malignant endocarditis has been observed by several writers. Probably the first case in which the mycotic nature was recog-

nized was one which occurred at the Montreal General Hospital and is reported in full in my lectures on malignant endocarditis. In addition to the ulceration of the valves there were four aneurisms of the arch, of which one was large and saccular, and three were not bigger than cherries. An extensive growth of micrococci was present.

A form of parasitic aneurism which occurs with great frequency in the mesenteric arteries of the horse is due to the development of the *strongylus armatus*.

Thoma has described a "traction" aneurism of the concavity of the arch at the point of insertion of the remnant of the ductus Botalli (Virchow's Archiv, Bd. 122).

And, lastly, there are cases in which without any definite cause there is a tendency to the development of aneurisms in various parts of the body. A remarkable instance of it in our profession was afforded by the brilliant Thomas King Chambers, who first had an aneurism in the left popliteal artery, eleven years subsequently an aneurism in the right leg which was cured by pressure, and finally aneurism of both carotid arteries.

Incidence of Aneurism.—At St. Bartholomew's Hospital during thirty years there were 631 cases of aneurism. In 468 the disease affected the aorta, in 80 the popliteal, in 21 the femoral, in 14 the subclavian, in 8 the carotid, in 6 the external iliac artery (Oswald A. Browne).

Aneurism of the Thoracic Aorta.

The causes which favor the development of arterio-sclerosis prevail in aortic aneurism, particularly alcohol, syphilis, and overwork. The greatest danger probably is in strong muscular men with commencing degenerative processes in the arteries (a consequence of syphilis or alcohol or a result of hereditary weakness of the arterial tissues), who during a sudden muscular exertion are liable to lacerate the media, the intima not yet being strengthened by compensatory thickening over a spot of mesarteritis. Aneurisms of the thoracic aorta vary greatly in size and shape. A majority of them are saccular. They may be small and situated just above the aortic ring. Others form large tumors which project externally and occupy a large portion of the upper thorax. Small sacs from the descending portion of the arch may compress the trachea or the bronchi. In the thoracic portion the sac may erode the vertebræ or grow into the pleural cavity and compress the lung. In some instances it grows through the ribs and appears in the back.

Symptoms.—The chief influence of an aneurism is manifested in what are known as pressure effects. In the absence of these the aneurisms attain a large size without producing symptoms or seriously interfering with the circulation. Indeed, a useful clinical subdivision as given by Bramwell is into three groups—aneurisms which are entirely latent and give no physical signs; aneurisms which present signs of intrathoracic pressure, although it is difficult or impossible to determine the nature of the lesion producing the pressure; and, lastly, aneurisms which produce dis-

tinct tumors with well-marked pressure symptoms and external signs. Broadbent makes another useful division into aneurism of symptoms and aneurism of physical signs. It is perhaps best to consider aneurisms of the aorta according to the situation of the tumor.

(*a*) *Aneurisms of the Ascending Portion of the Arch.*—When just above the sinuses of Valsalva they are often small and latent. The first symptom may be rupture, which usually takes place into the pericardium and causes instant death. Above the sinuses, along the convex border of the ascending part, aneurism frequently develops, and may grow to a large size, either passing out into the right pleura or forward, pointing at the second or third interspace, eroding the ribs and sternum, and producing large external tumors. In this situation the sac is liable, indeed, to compress the superior vena cava, causing engorgement of the vessels of the head and arm, sometimes compressing only the subclavian vein, and causing enlargement and œdema of the right arm. Perforation may take place into the superior vena cava, of which accident Pepper and Griffith have collected 29 cases. In rare instances, when the aneurism springs from the concave side of the vessels, the tumor may appear to the left of the sternum. Large aneurisms in this situation may cause much dislocation of the heart, pushing it down and to the left, and sometimes compressing the inferior vena cava, and causing swelling of the feet and ascites. The right recurrent laryngeal nerve is often pressed upon by these tumors. The innominate artery is rarely involved. Death commonly follows from rupture into the pericardium, the pleura, or into the superior cava; less commonly from rupture externally, sometimes from syncope.

(*b*) *Aneurisms of the Transverse Arch.*—The direction of their growth is most commonly backward, but they may grow forward, erode the sternum, and produce large tumors. The tumor presents in the middle line and to the right of the sternum much more often than to the left, which occurred in only 4 of 35 aneurisms in this situation (O. A. Browne). Even when small and producing no external tumor they may cause marked pressure signs in their growth backward toward the spine, involving the trachea and the œsophagus, and giving rise to cough, which is often of a paroxysmal character, and dysphagia. The left recurrent laryngeal is often involved in its course round the arch. A small aneurism from the lower or posterior wall of the arch may compress a bronchus, inducing bronchorrhœa, gradual bronchiectasy, and suppuration in the lung—a process which by no means infrequently causes death in aneurism, and a condition which at the Montreal General Hospital we were in the habit of terming aneurismal phthisis. Occasionally enormous aneurisms develop in this situation, and grow into both pleuræ, extending between the manubrium and the vertebræ; they may persist for years. The sac may be evident at the sternal notch. The innominate artery, less commonly the left carotid and subclavian, may be involved in the sac, and the radial or carotid pulse may be absent or retarded. Pressure on the sympathetic may at first cause dilatation and subsequently contraction of the pupil. Sometimes the thoracic duct is compressed.

The ascending and transverse portions of the arch are not infrequently

dilatation of the arch, and extending into the innominate artery, its entire length is a saccular dilatation, irregularly rounded in shape, two inches in diameter, from the upper and posterior portion of which are given off the subclavian and right carotid. This sacculation is filled with laminated clot. It includes also the left carotid, but not the left subclavian. The left carotid contains a thrombus to a point two inches above its origin, which occupies its entire lumen.

The left subclavian also contains a laminated thrombus, extending to a point an inch above its origin, and possibly further, totally occluding its lumen.

Lower two-thirds of right lung and apex of left lung tubercular with extensive cavity formation.

1946 Marshall Avenue.

THE IMPORTANCE OF EARLY EXTIRPATION OF THE TUBERCULOUS TESTICLE.

By W. D. Jones, M. D.

Devils Lake, N. D.

Until within the last few years comparatively little has been contributed to medical literature upon tuberculous diseases of the genito-urinary organs.

To Senn, the profession and humanity at large owe a debt of gratitude for his classical work upon this subject. While we are possessed of a comparatively clear pathology of genito-urinary diseases, clinically they are tinctured with diagnostic error and the unfortunate results of a treatment based upon a mistaken hypothesis.

Tuberculosis of the testicle is generally secondary to a tubercular process elsewhere within the organism; but it may occur de novo without any pre-existing tubercular condition. From a clinical view tubercular invasion of the testicle may be:

(1) Secondary to tuberculosis in some remote tissue; (2) secondary to tuberculosis in some contiguous structure; (3) secondary to some known traumatism; (4) secondary to simple or gonorrheal infection; (5) cases where there has been an infection atrium and the testicle is primarily involved and produces secondary disease of other structures; those of the genito-urinary system and lungs being most frequent.

So intimately related are the various organs constituting the genito-urinary tract through the blood vessels and lymphatics that it seems disease in one organ of this system must be followed by disease in another as a natural sequence.

It is very easy to conceive how a tubercular focus in a part remote from a testicle may lead to infection of this organ through the blood current; or a peripheral lesion in the vicinity of the genito-urinary tract may furnish an infection atrium through which the bacillus may gain access to the lymph stream and lead to infection of some part of this important system or distant organ.

When we recall the fact that by injecting the testicle with a fluid, we can fill the receptaculum chyli, and this, together with the rich lymphatic circulation of the testicle, we are in a position to appreciate the great danger a small and neglected tubercular focus of the testis may be to the human organism and the importance of early extirpation to protect adjacent organs and the system against disasterous results.

I will cite three case histories, one from literature and two from personal experience:

Case 1.—Service of Dr. R. W. Taylor, Charity Hospital, New York. J. R., age 40; American; bachelor; was admitted to Charity Hospital, Dec. 28, 1886. The family history on his father's side is excellent, but that of his mother is bad, since she, together with three brothers and three sisters, died of consumption prior to their 40th year. The patient says he had pains in his chest and shoulder blades, together with much expectoration fifteen years ago; that six years ago he had a hæmmorrhage, and that four years ago he was told by a physician he had consumption. He had never suffered from any venereal disease. In September, 1886, he fell from a high elevation but experienced no injury to his testes known to him. Shortly after this his left testis began to be very painful and to swell. The enlargement went steadily on accompanied by pain. The right testis became swollen and painful about Christmas, 1886, and on admission it was found larger than the left. Three weeks later an abscess was formed in the left testis which was by my direction opened by my house surgeon. About a fortnight later an abscess opened spontaneously in the right testis. The very weak and anæmic condition of the man, though the discharge of pus from the testis was great, lead me to defer operation until he had been built up by extra diet and tonics. On the 23rd day of February, I removed both testes under strict antisepsis. Healing was perfect at the end of a week.

Before the operation the patient expectorated a great deal; but since he has gradually improved in this respect; his health is much better and he has gained considerably in weight. In the sputum of this patient the tubercle bacillus was found.

Case 2.—D. B., age 36; American; married; good family history; had never had any venereal disease or serious illness until five years ago. Consulted me in April, 1899, for a tumor in his right side, which he attributed to enlargement of the liver. By close inquiry I learned he was suffering from frequent urination and discharge of pus, which he thought due to a cystitis, for

involved together, usually without the branches; the tumor grows upward, or upward and to the right.

(*c*) *Aneurisms of the Descending Portion of the Arch.*—The sac projects to the left and backward, and often erodes the vertebræ from the third to the sixth dorsal, causing great pain and sometimes compression of the spinal cord. Dysphagia is common. Pressure on the bronchi may induce bronchiectasy, with retention of secretions, and fever. A tumor may appear externally in the region of the scapula, and here attain an enormous size. Death not infrequently occurs from rupture into the pleura.

(*d*) *Aneurisms of the Descending Thoracic Aorta.*—The larger number occur close to the diaphragm, the sac lying upon or to the left of the bodies of the lower dorsal vertebræ, which are often eroded. The sac may reach a large size and form a very large tumor in the back.

Diagnosis and Physical Signs.—*Inspection.*—A good light is essential; cases are often overlooked owing to a hasty inspection. In many instances it is negative. On either side of the sternum there may be abnormal pulsation, due to dislocation of the heart, to deformity of the thorax, or to retraction of the lung. The aneurismal pulsation is usually above the level of the third rib and most commonly to the right of the sternum, either in the first or second interspace. It may be only a diffuse heaving impulse without any external tumor. Often the impulse is noticed only when the chest is looked at obliquely in a favorable light. When the innominate is involved the throbbing may pass into the neck or be apparent at the sternal notch. Posteriorly, when pulsation occurs, it is most commonly found to the left of the spine. An external tumor is present in many cases, projecting either through the upper part of the sternum or to the right, sometimes involving the sternum and costal cartilages on both sides, forming a swelling the size of a cocoa-nut or even larger. The skin is thin, often blood-stained, or it may have ruptured, exposing the laminæ of the sac. The apex beat may be much dislocated, particularly when the sac is large. It is more commonly a dislocation from pressure than from enlargement of the heart itself.

Palpation.—The area and degree of pulsation are best determined by palpation. When the aneurism is deep-seated and not apparent externally, the bimanual method should be used, one hand upon the spine and the other on the sternum. When the sac has perforated the chest wall the impulse is, as a rule, forcible, slow, heaving, and expansile. The resistance may be very great if there are thick laminæ beneath the skin; more rarely the sac is soft and fluctuating. The hand upon the sac, or on the region in which it is in contact with the chest wall, feels in many cases a diastolic shock, often of great intensity, which forms one of the valuable physical signs of aneurism. A systolic thrill is sometimes present, not so often in saccular aneurisms as in the dilatation of the arch. The pulsation may sometimes be felt in the suprasternal notch.

Percussion.—The small and deep-seated aneurisms are in this respect negative. In the larger tumors, as soon as the sac reaches the chest wall, there is produced an area of abnormal dulness, the position of which depends upon the part of the aorta affected. Aneurisms of the ascending

Hæmoptysis may occur as an early symptom in thoracic aneurism, or at any time in the course of the disease, and may be easily mistaken for that of tubercular, or valvular disease; these not fatal hæmorrhages indicate that ulceration and repair may go on without dangerous loss of blood, they may be frothy and streaked with blood, rusty, prune juice or pure blood, according as they may arise from soft granulations in the trachea, at the seat of pressure, ulceration into the trachea or a bronchus, or erosion of the lung tissue. Dysphagia is not a very common symptom, and, when present, the aneurism is usually found in the descending aorta; the difficulty in swallowing being due either to spasm or direct pressure. The objective symptoms or physical signs of thoracic aneurism are elicited by the usual methods of inspection, palpation, mensuration, percussion and auscultation. The proximity of the arch of the aorta to the sternum, at the second right sterno-costal articulation, gives rise to undue pulsation at that point, if there be even a moderate degree of dilatation. Pulsation may be felt in the suprasternal notch, or towards the innominate on the right side, or the left carotid on the left; and if within the limits of such pulsation, abnormal dullness on percussion is observed, with a visible heaving of the chest wall, it can be safely inferred that one has to deal with an aneurism or mediastinal tumor of some kind.

The careful mapping out of the area of dullness as to its continuance with, or separation from the heart dullness, is of importance.

If the aneurism is close to the heart, the dullness will be inseparable from that of the cardia, while a lesion of the transverse portion of the arch, or the innominate artery, will present a well defined separation of dullness. An aneurism extending backwards or downwards will be recognized with difficulty by percussion.

The impulse of an aneurism is expansile in character, and is usually better appreciated when considerable pressure is exerted by the hand; it corresponds with the ventricular systole, and is sometimes accompanied by a sense of vibration or thrill, and a second impulse or diastolic shock, coinciding with the second sound of the heart, and recoil of the vessels. The case which I have for your inspection shows how large a tumor (the size of an orange), involving, as it does, the innominate artery, can exist without showing any of this expansile impulse, because of the fact that the whole sac is occupied with a dense mass of fibrin. The only impulse seen and felt in this case was in the suprasternal notch, and to the right of the right sterno-mastoid. Pulsation sometimes occurs posteriorly in the left scapular region, and the apex beat of the heart may be dislocated, if the tumor be a large one. The presence of adventitious sounds mainly depends on the degree of thickness of the laminæ of fibrin within the sac. When the aneurism is near the heart, it may be impossible to differentiate between it and valvular disease by auscultation alone, but when murmurs are heard confined to an area of pulsation or dullness outside the heart area, and usually of increased intensity at that point, the existence of aneurism may be reasonably inferred.

The murmur is usually systolic. In some cases there is also a murmur corresponding with the recoil of the sac, with or without aortic insufficiency, and in rare cases the only murmur is diastolic. A very highly accentuated aortic second sound is frequently heard, the result of exaggerated recoil on healthy valves.

In the case of Mrs. D., there was no murmur audible over the aneurism, but a loud systolic murmur was heard with greatest intensity at the lower angle of the right scapula, and less distinctly down the spine in the lower two-thirds of the right interscapular region, this being the region occupied by that portion of the right lung involved in the tubercular process. Gairdner lays much stress on displacements of the trachea. He says the slightest lateral deviation of the trachea, at its lower part from a line drawn from the pomum adami to the precise middle of the suprasternal notch, is a sign of the most signal importance, and easy of recognition, as is also diminished mobility of the trachea in the act of swallowing. (Allbutt's System, Vol. VII, p. 393.) Tracheal tugging is a sign of much value in deep seated aneurisms. The trachea is put upon stretch by grasping the cricoid cartilage between the index finger and thumb and exerting upward pressure; if an aneurism is present, and adherent to the trachea at its bifurcation, or one or other bronchus, as it usually is in aneurism of the transverse portion of the arch, a distinct tugging downwards is experienced with each pulsation.

Hall, in the American Journal of the Medical Sciences, January, 1900, regards a tracheal diastolic shock as a more valuable sign than tracheal tugging, as the latter does not differentiate between aneurism and mediastinal tumor. The closure of the aortic valves transmits, through the aneurism to the trachea, a sharp impulse following the tracheal tug, by an interval the same as that between the apex beat and the closure of the aortic segments.

A constant and increasing weakening of the pulse on one side is important, and often conclusive; this weakening may go on to complete obliteration of one or the other radial pulse, and may extend to the carotids by gradual sealing up of the vessels by layers of fibrin within the sac. This feature was well marked in the case of my patient, there being entire absence of the radial pulse on both sides, and also of both caro-

tids. The modification in the arterial pulse is not always the result of occlusion of the vessel, but may be due to loss of elastic resistance in the vascular coats.

Pressure of an aneurism of the arch upon the superior vena cava or innominate vein, may give rise to the usual signs of venous obstruction, but this is more frequently seen in malignant or glandular tumors than in aneurism.

Changes in the pupil, the result of pressure on the sympathetic or the nerves of the cilio-spinal region, are frequently observed in aneurism of the ascending portion of the arch: simple irritation of communication, being followed by contraction.

The Argyll-Robertson pupil (where a contracted pupil is insensible to light, but subject to further contraction during the act of accommodation for near objects) is, according to Gairdner, "as characteristic of intrathoracic or aneurismal tumors, as it is of the other forms of spinal myosis." Unilateral pallor, congestion or sweating, are also frequently observed as the result of such pressure. Pressure on the thoracic duct is attended with the usual symptoms.

The employment of the X-ray in the diagnosis of thoracic aneurism has received considerable attention, and frequently successful results have been noted, not only as to the extent and situation of the tumor, but also where the diagnosis has been made by this means alone. An attempt was made in the case of Mrs. D. to secure a radiograph, and while direct vision by the fluoroscope gave a fairly intelligible view of the growth, unfortunately the extreme restlessness of the patient interfered with the success of the experiment.

Case. Mrs. D., age 42, admitted to the City and County Hospital Feb. 7th, 1900. Her father, one sister and a maternal aunt died of consumption. She was occupied as a housemaid up to age of 22, at which time she married. She had six miscarriages, and only one child at full term. Healthy up to eight years ago, when she began to have attacks of fainting and spells of dizziness, slight hacking cough and a feeling of tightness about the chest. Two years ago she was operated upon by Dr. Stone, of this city, for laceration of the cervix. At this time unusual pulsation was observed in the suprasternal notch, and complete absence of the pulse in both radials was also noted. The anæsthetic (ether) was well borne.

During the six months previous to her admission to hospital, she failed steadily, her attacks of fainting and dizziness which persisted since their onset, became more and more frequent, she lost flesh rapidly, coughed and had bloody expectoration, and profuse night sweats. Frequent examination of the sputum made during this period failed to reveal the presence of the tubercle bacillus. On admission her condition suggested an advanced stage of consumption. On examination there was entire absence of pulsation in both upper extremities, as well as in the carotids, and temporal vessels. Pulsation was, however, distinct in the abdominal aorta, and throughout the lower extremities. The suprasternal notch was occupied by a pulsating mass extending three-quarters of an inch above the upper margin of the sternum, one inch to the right and three-quarters of an inch to the left of the midsternal line. Pulsation not expansile, but merely an upward systolic impulse. Cardiac impulse barely visible and no visible pulsation or heaving of the chest wall. There was dullness over the visible mass, and extending downwards to the upper border of the second rib. Laterally the mass could not be limited by percussion, owing to general dullness over the entire right lung, and the left apex. Heart sounds weak but clear; no adventitious sounds audible over the mass, but at the lower angle of the right scapula, a loud systolic murmur was heard through solidified lung. This murmur was also heard in the right interscapular space, but not so loudly. Tracheal tugging and the so called diastolic shock were not observed. Left pupil slightly dilated, both pupils reacted to light and accommodation, but sluggishly, slight dysphagia. Cough and alteration of voice more characteristic of a tubercular condition than aneurism. Examination by means of the laryngoscope was not attempted owing to the weakness of the patient. Death by exhaustion.

Autopsy. On removal of sternum, a solid mass was exposed, size of a small orange, the center of which corresponded to a line drawn across the middle of the sternal ends of clavicles, and extending three quarters of an inch more to the right. No erosions of sternum or clavicle. Under surface of right clavicle presented a hollow depression close to the articulation, deep enough to fit the end of finger. No vertebral erosions.

Heart small; slight thickening of both mitral leaflets. At the base of the posterior leaflet on the ventricular side, there is a small patch of atheroma, muscle firm with some infiltration of fat, coronary arteries rigid and atheromatous, nothing special on right sode. Aortic valves slightly thickened, margins free, and valves free from atheroma. Aorta immediately above the valves is markedly atheromatous, and openings into coronary arteries are contracted and surrounded by patches of atheroma. The lining of the aorta is rough and atheromatous, with here and there deposits of lime salts. Atheromatous changes increase as the arch is reached, where it is present in a most marked degree. No dilatation of first portion of aorta. There is uniform

W.O.

I will be very glad to send you this specimen if you would like to have it

W.A.

ORIGINAL ARTICLES.

DIAGNOSIS OF THORACIC ANEURISM.

By Andrew Henderson, M. D.

The [illegible] the [illegible] by which the [illegible] value in [illegible] of [illegible] may prove of much value in [illegible] times [illegible] may prove of much value in diagnosis. Cardiac syncope and irregularity, such as is observed in angina pectoris, is found exceptionally in aortic aneurism, and when it occurs it usually implies that the sac arises [illegible] such aneurisms usually un

ORIGINAL ARTICLES.

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By Andrew Henderson, M. D.

St. Paul.

As the early recognition of aneurism is of the greatest importance to the patient, and interest to the physician, it behooves the latter to employ his mental faculties to the utmost in all cases presenting the least suspicious element, long before the physical signs or symptoms render the condition obvious.

Sir Wm. J. Gairdner, in his article in Allbutt's System of Medicine, presents the diagnosis of aneurism of the aorta on three distinct lines, all of which may come into play in various degrees.

1. The ætiological factor, involving questions of predisposition.

2. The symptomatic factor, founded on the active manifestations of illness.

3. The factor of physical diagnosis proper, or the objective phenomena indicating tumor.

In considering the ætiological diagnosis of aneurism, the fact is to be borne in mind that this condition is rarely to be found in youth, adolescence and early manhood and womanhood, its period of most frequent occurrence being between the ages of 30 and 50, the age depending more upon the condition of the arteries.

A history of alcoholism, overwork or syphilis in a person of such an age, subjected to a sudden strain or severe muscular exertion, should always excite suspicion.

The symptomatology of thoracic aneurism depends in the main on the effects of pressure. Pain, while not always present, is often the first and most noted symptom; it is very variable in its character and duration. When due to pressure on sensory nerves, it is usually sharp, paroxysmal, and subject to remissions, and when it is the result of erosion of the bony structures with which the aneurism comes in contact, it is dull, gnawing and constant; some times it resembles closely that of angina pectoris. The importance of pain as an early symptom of aneurism is emphasized by Gairdner. He holds it to be a good practical rule, that whenever you encounter in your experience a case of obstinate or frequently recurring pain, such as might constructively be due to pressure upon nerves or upon solid parts, and such as is not fairly in accordance with some disease known to exist in the thorax or abdomen, the suspicion, at least, of an aneurism ought, in all cases, to arise. The therapeutic test of large doses of iodide of potassium, by which the pain is oftentimes alleviated, may prove of much value in diagnosis. Cardiac syncope and irregularity, such as is observed in angina pectoris, is found exceptionally in aortic aneurism, and when it does occur it usually implies that the sac arises in the first part of the arch and very close to the heart. The structure of the heart is usually unaffected, especially where the transverse portion of the arch is involved: this is shown in the case which I am presenting. It will be seen that the heart is small and with the exception of only a slight thickening of the mitral leaflets its structure is unaltered.

Perhaps the most frequent of the early symptoms is cough, and in some cases this is so characteristic that a diagnosis may be guessed at from this symptom alone. The cough has been described as hoarse, clanging, laryngeal and brassy; it is a cough with an imperfectly closed glottis, says Gairdner, "and when found in association with even the slightest degree of stridor or dyspnœa, is as nearly absolutely distinctive of thoracic tumor or aneurism as anything apart from its patent physical signs can possibly be." (Allbutts' system of Med., Vol. VI, p. 377.) Certain alterations in the voice taken in connection with other symptoms already mentioned, may reasonably give rise to suspicion of aneurism. The voice may be husky and coarse, at times breaking into a falsetto on attempting to speak loudly, and there may be complete aphonia, such varieties of voice being due to chronic laryngitis, œdema of the glottis from venous stasis, or paralysis of the vocal cords.

Sir Felix Semon, in his article on "Laryngeal Neuroses in Allbutts' System of Medicine, says "that in all progressive organic lesions of the centres or trunks of the motor laryngeal nerves, the abductors of the vocal cords succomb much earlier than the adductors, that paralysis of the adductors is almost invariably bilateral, and due to functional disorders, while unilateral paralysis of the abductors almost always indicates organic disease, and as unilateral abductor paralysis is not necessarily attended with any vocal or respiratory disturbance, it can only be recognized by the laryngscope, which should be invariably employed where the remotest presumption of thoracic tumor can arise." Cases of violently suffocative paroxysmal attacks are more likely due to spasm of the adductors, arising from reflex irritation through the vagus. (Gairdner.)

arch grow forward and to the right, producing dulness on one side of the manubrium; those from the transverse arch produce dulness in the middle line, extending toward the left of the sternum, while aneurisms of the descending portion most commonly produce dulness in the left interscapular and scapular regions. The percussion note is flat and gives a feeling of increased resistance.

Auscultation.—Adventitious sounds are not always to be heard. Even in a large sac there may be no murmur. Much depends upon the thickness of the laminæ of fibrin. An important sign, particularly if heard over a dull region, is a ringing, accentuated second sound, a phenomenon rarely missed in large aneurisms of the aortic arch. A systolic murmur may be present; sometimes a double murmur, in which case the diastolic *bruit* is usually due to associated aortic insufficiency. The systolic murmur alone is of little moment in the diagnosis of an aneurismal sac. With the single stethoscope the shock of the impulse with the first sound is sometimes very marked.

Among other physical signs of importance are slowing of the pulse in the arteries beyond the aneurism, or in those involved in the sac. There may, for instance, be a marked difference between the right and left radial, both in volume and time. A physical sign of large thoracic aneurism, which I have not seen referred to, is obliteration of the pulse in the abdominal aorta and its branches. My attention was called to this in a patient who was stated to have aortic insufficiency. There was a well-marked diastolic murmur, but in the femorals and in the aorta I was surprised to find no trace of pulsation, and not the slightest throbbing in the abdominal aorta or in the peripheral arteries of the leg. The circulation was, however, unimpaired in them and there was no dilatation of the veins. Attracted by this, I then made a careful examination of the patient's back, when the circumstance was discovered, which neither the patient himself nor any of his physicians had noticed, that he had a very large area of pulsation in the left scapular region. The sac probably was large enough to act as a reservoir annihilating the ventricular systole, and converting the intermittent into a continuous stream.

The *tracheal tugging*, a valuable sign in deep-seated aneurisms, was described by Surgeon-Major Oliver, and was specially studied by my colleagues Ross and MacDonnell * at the Montreal General Hospital. Oliver gives the following directions: "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurism exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." On several occasions I have known this to be a sign of great value in the diagnosis of deep-seated aneurisms. I have never felt it in tumors, or in the extreme dynamic dilatation of aortic insufficiency.

Occasionally a systolic murmur may be heard in the trachea, as pointed

* London Lancet, 1891.

It may be visible in the thyroid cartilage.

In some instances the injection is at the
base of the adductor muscle
of the thigh. (Case no E. 11.1.41.)

out by David Drummond, or even at the patient's mouth, when opened. This is either the sound conveyed from the sac, or is produced by the air as it is driven out of the wind-pipe during the systole.

An important but variable feature in thoracic aneurism is *pain*, which is particularly marked in deep-seated tumors. It is usually paroxysmal, sharp, and lancinating, often very severe when the tumor is eroding the vertebræ, or perforating the chest wall. In the latter case, after perforation the pain may cease. Anginal attacks are not uncommon, particularly in aneurisms at the root of the aorta. Frequently the pain radiates down the left arm or up the neck, sometimes along the upper intercostal nerves. *Cough* results either from the direct pressure on the wind-pipe, or is associated with bronchitis. The expectoration in these instances is abundant, thin, and watery; subsequently it becomes thick and turbid. Paroxysmal cough of a peculiar brazen, ringing character is a characteristic symptom in some cases, particularly when there is pressure on the recurrent laryngeal nerves, or the cough may have a peculiar wheezy quality—the "goose cough."

Dyspnœa, which is common in cases of aneurism of the transverse portion, is not necessarily associated with pressure on the recurrent laryngeal nerves, but may be due directly to compression of the trachea or the left bronchus. It may occur with marked stridor. Loss of voice and hoarseness are consequences of pressure on the recurrent laryngeal, usually the left, inducing either a spasm in the muscles of the left vocal cord or paralysis.

Paralysis of an abductor on one side may be present without any symptoms. It is more particularly, as Semon states, when the paralytic contractures supervene that the attention is called to laryngeal symptoms.

Hæmorrhage in thoracic aneurism may come from (*a*) the soft granulations in the trachea at the point of compression, in which case the sputa are blood-tinged, but large quantities of blood are not lost; (*b*) from rupture of the sac into the trachea or bronchi; (*c*) from perforation into the lung or erosion of the lung tissue. The bleeding may be profuse, rapidly proving fatal, and is a common cause of death. It may persist for weeks or months, in which case it is simply hæmorrhagic weeping through the sac, which is exposed in the trachea. In some instances, even after a very profuse hæmorrhage, the patient recovers and may live for years. A man with well-marked thoracic aneurism, whom I showed to my class at the University of Pennsylvania and who had had several brisk hæmorrhages, died four years after, having in the meantime enjoyed average health. Death from hæmorrhage is relatively more common in aneurism of the third portion of the arch and of the descending aorta.

Difficulty of swallowing is a comparatively rare symptom, and may be due either to spasm or to direct compression. The sound should never be passed in these cases, as the œsophagus may be almost eroded and a perforation may be made.

Heart Symptoms.—Pain has been referred to; it is often anginal in character, and is most common when the root of the aorta is involved. The heart is hypertrophied in less than one half the cases. The aortic valves

are sometimes incompetent, either from disease of the segments or from stretching of the aortic ring.

Among other signs and symptoms, venous compression, which has already been mentioned, may involve one subclavian or the superior vena cava. A curious phenomenon in intrathoracic aneurism is the clubbing of the fingers and incurving of the nails of one hand, of which two examples have been under my care, in both without any special distention or signs of venous engorgement. Tumors of the arch may involve the pulmonary artery, producing compression, or in some instances adhesion of the pulmonary segments and insufficiency of the valve; or the sac may rupture into the artery, an accident which happened in two of my cases, producing instantaneous death.

Pressure on the sympathetic is particularly liable to occur in growths from the ascending portion of the arch. Either the upper dorsal or the lower cervical ganglion is involved. The symptoms are variable. If the nerve is simply irritated, there is stimulation of the vaso-dilator fibres and dilatation of the pupil. With this may be associated pallor of the same side of the face. On the other hand, destruction of the cilio-spinal branches causes paralysis of the dilator fibres, in consequence of which the iris contracts, the vessels on the side of the head dilate, causing congestion, and in some instances unilateral sweating. It is much more common to see the pupillary symptoms alone than in combination either with pallor, redness, or sweating.

The clinical picture of aneurism of the aorta is extremely varied. Many cases present characteristic symptoms and no physical signs, while others have well-marked physical signs and no symptoms. As Broadbent remarks, the aneurism of *physical signs* springs from the ascending portion of the aorta; the aneurism of *symptoms* grows from the transverse arch.

Aneurism of the aorta may be confounded with: (*a*) The violent throbbing impulse of the arch in aortic insufficiency. I have already referred to a case of this kind in which the diagnosis of aneurism was made by several good observers.

(*b*) *Simple Dynamic Pulsation.*—No instance of this, which is common in the abdominal aorta, has ever come under my notice. One which came under the care of William Murray and Bramwell presented, without any pain or pressure symptoms, pulsation and dulness over the aorta. The condition gradually disappeared and was thought to be neurotic.

(*c*) Dislocation of the heart in curvature of the spine may cause great displacement of the aorta, so that it has been known to pulsate forcibly to the right of the sternum.

(*d*) *Solid Tumors.*—When the tumor projects externally and pulsates the difficulty may be considerable. In tumor the heaving, *expansile* pulsation is absent, and there is not that sense of force and power which is so striking in the throbbing of a perforating aneurism. There is not to be felt as in aortic aneurism the shock of the heart-sounds, particularly the diastolic shock. Auscultatory sounds are less definite, as large aneurisms may occur without murmur; and, on the other hand, murmurs may be heard over tumors. The greatest difficulty is in the deep-seated thoracic

tumors, and here the diagnosis may be impossible. I have already referred to the case which was regarded by Skoda as aneurism and by Oppolzer as tumor. The physical signs may be indefinite. The ringing aortic second sound is of great importance and is rarely, if ever, heard over tumor. Tracheal tugging is here a valuable sign. Pressure phenomena are less common in tumor, whereas pain is more frequent. The general appearance of the patient in aneurism is much better than in tumor, in which there may be cachexia and enlargement of the glands in the axilla or in the neck. Healthy, strong males who have worked hard and have had syphilis are the most common subjects of aneurism. Occasionally cancer of the œsophagus may simulate aneurism, producing pressure on the left bronchus, and in one instance at the Philadelphia Hospital, with a husky, brazen cough, the symptoms were very suggestive.

(*e*) *Pulsating Pleurisy.*—In cases of *empyema necessitatis*, if the projecting tumor is in the neighborhood of the heart and pulsates, the condition may readily be mistaken for aneurism. The absence of the heaving, firm distention and of the diastolic shock would, together with the history and the existence of pleural effusion, determine the nature of the case. If necessary, puncture may be made with a fine hypodermic needle. In a majority of the cases of pulsating pleurisy the throbbing is diffuse and widespread, moving the whole side.

Prognosis.—The outlook in thoracic aneurism is always grave. Life may be prolonged for some years, but the patients are in constant jeopardy. Spontaneous cure is not very infrequent in the small sacculated tumors of the ascending and thoracic portions. The cavity becomes filled with laminæ of firm fibrin, which become more and more dense and hard, the sac shrinks considerably, and finally lime salts are deposited in the old fibrin. The laminæ of fibrin may be on a level with the lumen of the vessel, causing complete obliteration of the sac. The cases which rupture externally, as a rule run a rapid course, although to this there are exceptions; the sac may contract, become firm and hard, and the patient may live for five, or even, as in a case mentioned by Balfour, for ten years. The cases which have lasted longest in my experience have been those in which a saccular aneurism has projected from the ascending arch. One patient in Montreal had been known to have aneurism for eleven years. The aneurism may be enormous, occupying a large area of the chest, and yet life be prolonged for many years, as in the case mentioned as under the care of Skoda and Oppolzer. One of the most remarkable instances is the case of dissecting aneurism reported by Graham. The patient was invalided after the Crimean War with aneurism of the aorta, and for years was under the observation of J. H. Richardson, of Toronto, under whose care he died in 1885. The autopsy showed a healed aneurism of the arch, with a dissecting aneurism extending the whole length of the aorta, which formed a double tube.

Treatment.—In a large proportion of the cases this can only be palliative. Still in every instance measures should be taken which are known to promote clotting and consolidation within the sac. In any large series of cured aneurisms a considerable majority of the patients have not been

known to be subjects of the disease, but the obliterated sac has been found accidentally at the post mortem.

The most satisfactory plan in early cases, when it can be carried out thoroughly, is that advised by the late Mr. Tufnell, of Dublin, the essentials of which are rest and a restricted diet. Rest is essential and should, as far as possible, be absolute. The reduction of the daily number of heart-beats, when a patient is recumbent and makes no exertion whatever, amounts to many thousands, and is one of the principal advantages of this plan. Mental quiet should also be enjoined. The diet advised by Tufnell is extremely rigid—for breakfast, 2 ounces of bread and butter and 2 ounces of milk; for dinner, 2 or 3 ounces of meat and 3 or 4 ounces of milk or claret; for supper, 2 ounces of bread and 2 ounces of milk. This low diet diminishes the blood-volume and is thought also to render the blood more fibrinous. It reduces greatly the blood-pressure within the sac, in this manner favoring coagulation. This treatment should be pursued for several months, but, except in persons of a good deal of mental stamina, it is impossible to carry it out for more than a few weeks at a time. It is a form of treatment adapted only for the saccular form of aneurism, and in cases of large sacs communicating with the aorta by a comparatively small orifice the chances of consolidation are fairly good. Unquestionably rest and the restriction of the liquids are the important parts of the treatment, and a greater variety and quantity of food may be allowed with advantage. If this plan cannot be thoroughly carried out, the patient should at any rate be advised to live a very quiet life, moving about with deliberation and avoiding all sudden mental or bodily excitement. The bowels should be kept regular, and constipation and straining should be carefully avoided. Of medicines, iodide of potassium, as advised by Balfour, is of great value. It may be given in doses of from 10 to 15 or 20 grains three times a day. Larger doses are not necessary. The mode of action is not well understood. It may act by increasing the secretions and so inspissating the blood, by lowering the blood-pressure, or, as Balfour thinks, by causing thickening and contraction of the sac. The most striking effect of the iodide in my experience has been the relief of the pain. The evidence is not conclusive that the syphilitic cases are more benefited by it than the non-syphilitic. All these measures have little value unless the sac is of a suitable form and size. The large tumors with wide mouths communicating with the ascending portion of the aorta may be treated on the most approved plans for months without the slightest influence other than reduction in the intensity of the throbbing. A patient with a tumor projecting into the right pleura remained on the most rigid Tufnell treatment for more than one hundred days, during which time he also took iodide of potassium faithfully. The pulsations were greatly reduced and the area of dulness diminished, and we congratulated ourselves that the sac was probably consolidating. Sudden death followed rupture into the pleura, and the sac contained only fluid blood, not a shred of fibrin. In cases in which the tumor is large, or in which there seems to be very little prospect of consolidation, it is perhaps better to advise a man to go on quietly with his occupation, avoiding excitement and worry. Our

profession has offered many examples of good work, thoroughly and conscientiously carried out, by men with aneurism of the aorta, who wisely, I think, preferred, as did the late Hilton Fagge, to die in harness.

Surgical Measures.—In a few cases consolidation may be promoted in the sac by the introduction of a foreign body, such as wire, horse-hair, or by the combination of wiring and electrolysis. Moore, in 1864, first wired a sac, putting in ~~28~~ feet of fine wire. Death occurred on the fifth day. Corradi proposed the combined method of wiring with electrolysis, which was first used by Burresi in 1879. His patient lived for three and a half months. Horse-hair, watch-spring wire, catgut, and Florence silk have been used. Hunner has collected for me the statistics of Moore's method (wiring), of which there were 13 cases, 8 of thoracic aneurism, all fatal; 5 aneurisms of the abdominal aorta, 2 of which were successful. Of 10 cases treated by wiring and electrolysis (Corradi's method), all were thoracic; of these, the cases of Kerr, Rosenstirn, D. D. Stewart, and Hershey, all American cases, were successful. The most favorable cases are those in which the aneurism is sacculated, but this is a point not easily determined, and often from a sac particularly favorable for wiring there may be secondary projections of great thinness. ~~In a case~~ of abdominal aneurism recently operated upon by Halsted all the conditions were very favorable, and the ~~man seemed doing very well~~ when sudden death occurred on the third day from rupture of a small projection of the sac through the diaphragm ~~into the pleura~~.

Other Symptoms requiring Treatment.—Pressure on veins causing engorgement, particularly of the head and arms, is sometimes promptly relieved by free venesection, and at any time during the course of a thoracic aneurism, if attacks of dyspnœa with lividity supervene, bleeding may be resorted to with great benefit. It has the advantage also of promptly checking the pain, for which symptom, as already mentioned, the iodide of potassium often gives relief. In the final stages morphia is, as a rule, necessary. Dyspnœa, if associated with cyanosis, is best relieved by bleeding. Chloroform inhalations may be necessary. The question sometimes comes up with reference to tracheotomy in these cases of urgent dyspnœa. If it can be shown by laryngoscopic examination that it is due to bilateral abductor paralysis the trachea may be opened, but this is extremely rare, and in nearly every instance the urgent dyspnœa is caused by pressure about the bifurcation. When the sac appears etxernally and grows large, an ice-cap may be applied upon it, or a belladonna plaster to allay the pain. In some instances an elastic support may be used with advantage, and I saw a physician with an enormous external aneurism in the right mammary region who for many months had obtained great relief by the elastic support, passing over the shoulder and under the arm of the opposite side.

Digitalis, ergot, aconite, and veratrum viride are rarely, if ever, of service in thoracic aneurism.

Aneurism of the Abdominal Aorta.

The sac is most common just below the diaphragm in the neighborhood of the cœliac axis. This variety is rare in comparison with thoracic aneurism. Of the 468 cases of aortic aneurism at St. Bartholomew's Hospital, 23 involved the abdominal aorta. The tumor may be fusiform or sacculated, and it is sometimes multiple. Projecting backward, it erodes the vertebræ and may cause numbness and tingling in the legs and finally paraplegia, or it may pass into the thorax and burst into the pleura. More commonly the sac is on the anterior wall and projects forward as a definite tumor, which may be either in the middle line or a little to the left. The tumor may project in the epigastric region (which is most common), in the left hypochondrium, in the left flank, or in the lumbar region. When high up beneath the pillar of the diaphragm it may attain considerable size without being very apparent on palpation.

The symptoms are chiefly pain, very often of a cardialgic nature, passing round to the sides or localized in the back, and gastric symptoms, particularly vomiting. Retardation of the pulse in the femoral is a very common symptom.

Diagnosis and Physical Signs.—Inspection may show marked pulsation in the epigastric region, sometimes a definite tumor. A thrill is not uncommon. The pulsation is forcible, expansile, and sometimes double when the sac is large and in contact with the pericardium. On palpation a *definite tumor can be felt.* If large, there is some degree of dulness on percussion which usually merges with that of the left lobe of the liver. On auscultation, a systolic murmur is, as a rule, audible, and is sometimes best heard at the back. A diastolic murmur is occasionally present, usually very soft in quality. One of the commonest of clinical errors is to mistake a throbbing aorta for an aneurism. It is to be remembered that no pulsation, however forcible, or the presence of a thrill or a systolic murmur justifies the diagnosis of abdominal aneurism unless there is a *definite tumor which can be grasped and which has an expansile pulsation.* Attention to this rule will save many errors. The throbbing aorta —the "preternatural pulsation in the epigastrium," as Allan Burns calls it—is met with in all neurasthenic conditions, particularly in women. In anæmia, particularly in some instances of traumatic anæmia, the throbbing may be very great. In the case of a large, stout man with severe hæmorrhages from a duodenal ulcer the throbbing of the abdominal aorta not only shook violently the whole abdomen, but communicated a pulsation to the bed, the shock of which was distinctly perceptible to any one sitting upon it. Very frequently a tumor of the pylorus, of the pancreas, or of the left lobe of the liver is lifted with each impulse of the aorta and may be confounded with aneurism. The absence of the forcible expansile impulse and the examination in the knee-elbow position, in which the tumor, as a rule, falls forward, and the pulsation is not then communicated, suffice for differentiation. The tumor of abdominal aneurism, though usually fixed, may be very freely movable.

The outlook in abdominal aneurism is bad. A few cases heal spon-

1 Henry Morris has collected 21 instances (Lancet, Oct. 6, 1900), 12 of which arose from injury. Many of them were false. Pulsation and a bruit are not always present. Four cases were operated upon; three recovered. In a case of Keen's the tumor and the kidney were removed together.

taneously. Death may result from (*a*) complete obliteration of the lumen by clots; (*b*) compression paraplegia; (*c*) rupture (which is almost the rule) either into the pleura, retroperitoneal tissues, peritonæum or the intestines, very commonly the duodenum; (*d*) by embolism of the superior mesenteric artery, producing infarction of the intestines.

The *treatment* is such as already advised in thoracic aneurism. When the aneurism is low down pressure has been successfully applied in a case by Murray, of Newcastle. It must be kept up for many hours under chloroform. The plan is not without risk, as patients have died from bruising and injury of the sac.

Aneurism of the Branches of the Abdominal Aorta.

The *cœliac axis* is itself not infrequently involved in aneurism of the first portion of the abdominal aorta. Of its branches, the *splenic artery* is occasionally the seat of aneurism. This rarely causes a tumor large enough to be felt; sometimes, however, the tumor is of large size. I have reported a case in a man, aged thirty, who had an illness of several months' duration, severe epigastric pain and vomiting, which led his physicians in New York to diagnose gastric ulcer. There was a deep-seated tumor in the left hypochondriac region, the dulness of which merged with that of the spleen. There was no pulsation, but it was thought on one occasion that a *bruit* was heard. The chief symptoms while under observation were vomiting, severe epigastric pain, occasional hæmatemesis, and finally severe hæmorrhage from the bowels. An aneurism of the splenic artery the size of a cocoa-nut was situated between the stomach above and the transverse colon below, and extended to the left as far as the level of the navel. The sac contained densely laminated fibrin. It had perforated the colon. I have twice seen small aneurisms on the splenic artery. Of 39 instances of aneurism on the branches of the abdominal aorta collected by Lebert, 10 were of the splenic artery.

Aneurism of the *hepatic artery* is very rare, and there are only 10 or 12 cases on record. The symptoms are extremely indefinite; the condition could rarely be diagnosed. In the case reported by Ross and myself, a man aged twenty-one had the symptoms of pyæmia. The liver was greatly enlarged, weighed nearly 5,000 grammes, and presented innumerable small abscesses. An oval aneurism, half the size of a small lemon, involved the right and part of the left branches. In J. B. S. Jackson's * case the aneurism perforated the hepatic duct.

A few cases of aneurism of the *superior mesenteric artery* are on record. The diagnosis is scarcely possible. Plugging of the branches or of the main stem may cause the symptoms of infarction of the bowels which have already been considered.

Small aneurisms of the *renal artery* are not very uncommon. Large tumors are rare. The sac may rupture and give rise to extensive retroperitoneal hæmorrhage.

* Medical Magazine, 1834, iii.

Arterio-venous Aneurism.

In this form there is abnormal communication between an artery and a vein. When a tumor lies between the two it is known as varicose aneurism; when there is a direct communication without tumor the vein is chiefly distended and the condition is known as aneurismal varix.

An aneurism of the ascending portion of the arch may open directly into the vena cava. Twenty-nine cases of this lesion have been analyzed by Pepper and Griffith. Cyanosis, œdema, and great distention of the veins of the upper part of the body are the most frequent symptoms, and develop, as a rule, with suddenness. Of the physical signs a thrill is present in some cases. A continuous murmur with systolic intensification is of great diagnostic value. In a recent case, after the existence for some time of pressure symptoms, intense cyanosis developed with engorgement of the veins of the head and arms. Over the aortic region there was a loud continuous murmur with systolic intensification.

A majority of the cases of arterio-venous aneurism and of aneurismal varix result from the accidental opening of an artery and vein as in venesection, and are met with at the bend of the elbow or sometimes in the temporal region. The condition may persist for years without causing any trouble. Pulsation, a loud thrill, and a continuous humming murmur are usually present.

Congenital Aneurism.

In consequence of failure of proper development of the elastic coat in many places in the arterial system, multiple aneurisms may develop. In the well-known case described by Kussmaul and Maier, upon many of the medium-sized arteries there were nodular prominences, which consisted of thickening of the intima and infiltration of the adventitia and of the media, with a nuclear growth which in places looked quite sarcomatous. They called it a case of *periarteritis nodosa*, and Eppinger holds that it belongs to the category which he makes of congenital aneurism. As many as 63 aneurismal tumors have been found in one case. In the smaller branches, such as the coronary and the mesenteric arteries or in the pulmonary arteries, there may be numerous elongated or saccular aneurisms varying in size from a cherry to a hazel-nut. These are true aneurismal dilatations, and, according to Eppinger's careful study, the wall consists of the intima and the adventitia, the elastic lamina having disappeared. The condition has been met with in children. Some of the cases, however, have been in adults; but the term as applied by Eppinger expresses, and probably correctly, the deep-seated fundamental error in development which must be at the basis of this condition. A favorite situation is in the coronary arteries; a case has been reported by Gee in a boy of seven.

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Turner reports the statistical results of both methods up to October, 1900. With Moore's method (wiring) 14 cases were treated, 8 of thoracic aneurism, all fatal; 6 aneurisms of the abdominal aorta, 3 of which were successful. Of 23 cases treated by wiring and electrolysis (Moore-Corradi method) 17 were thoracic and 6 abdominal. The thoracic cases of Rosenstein, Steward, and Kerr, and the abdominal cases of Noble and Finney (Case V) were successful. Eight of these 23 cases resulted in amelioration of symptoms and prolongation of life.

The most favorable cases are those in which the amputation is accelerated, but this is a point not easily determined, and often from a case particularly favorable for waiting there may be secondary projections of great thinness.

37

The sudden filling by clot of an aneurism of the coeliac axis or of the superior mesenteric artery may result fatally from infarct of the intestine.

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SECTION VIII.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

I. ANÆMIA.

Anæmia may be defined as a reduction in the amount of the blood as a whole or of its corpuscles, or of certain of its more important constituents, such as albumin and hæmoglobin. The condition may be general or local. The former alone we are here considering. It is interesting to note, however, that the pallor, particularly of the face, which is one of the most striking symptoms of anæmia, is just as characteristic of local anæmia due to fright or to nausea. There are persons persistently pale without actual anæmia in whom the condition may be due to inherited peculiarities.

Our knowledge is not yet sufficiently advanced to classify satisfactorily the various forms of anæmia. The following provisional grouping may be made: (1) Secondary or symptomatic anæmia; (2) primary, essential, or cytogenic anæmia.

Secondary Anæmia.

Under this division comes a large proportion of all cases. The following are the most important groups, based on the etiology:

(1) *Anæmia from hæmorrhage*, either traumatic or spontaneous. The loss of blood may be rapid, as in lesions of large vessels, in injury or in rupture of aneurisms, in cases of ulcer of the stomach or duodenum, or in post-partum hæmorrhage. If the loss is excessive, death results from lowering of the arterial pressure. In sudden profuse hæmorrhage the loss of 3 or 4 pounds of blood may prove fatal. In the rupture of an aneurism into the pleura the loss of blood may amount to $7\frac{1}{2}$ pounds, the largest quantity I have known to be shed into one cavity. In a case of hæmatemesis the patient lost over 10 pounds by measurement in one week and yet recovered from the immediate effects. Even after very severe hæmorrhage the number of red blood-corpuscles is not reduced so greatly as in forms of idiopathic anæmia. Thus in one case just mentioned, at the termination of the week of bleeding there were nearly 1,390,000 red blood-corpuscles to the cubic millimetre. The process of regeneration goes on with great rapidity, and in some "bleeders" a week or ten days suffice

Hospital services at Christchurch Public.

A series of cases has been described in which the lesions are small aneurisms on the arteries of the cerebellum and viscera. The first case was reported by Leopold and Meier [illegible], and three other have been described. A fifth case, attended clinically by the authors of this address, has remained in my care. No autopsy was permitted, but the evidence seems built to be accepted until further cases. The case is reported by [illegible] (a modified lesion)

[illegible]; the other they are [illegible] set.

The cases [illegible] which usually is one of acute delirium [illegible] of 37 and 39; the course from start to termination. The patient complains of weakness. The arteries correspond with the lesions. There are seizures in the vessels accompanied with pain, dizziness and sometimes paralysis and stupor. The writer has studied it for eleven years. An account [illegible] pain; there is loss of appetite, thirst, vomiting, [illegible] of disease. The disease is fatal at first, but the [illegible] have to succumb, while the other system recovers. In one case the [illegible] was 37 and 39; in cells 1,704,002. The [illegible] pain [illegible] after 24 per cent were [illegible]. The writer is [illegible] of the specific cruelty, also [illegible] [illegible] See [illegible]

VIII.

OD AND DUCTLESS DS.

MIA.

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Anæmia.

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to re-establish the normal amount. The watery and saline constituents of the blood are readily restored by absorption from the gastro-intestinal tract. The albuminous elements also are quickly renewed, but it may take weeks or months for the corpuscles to reach the normal standard. The

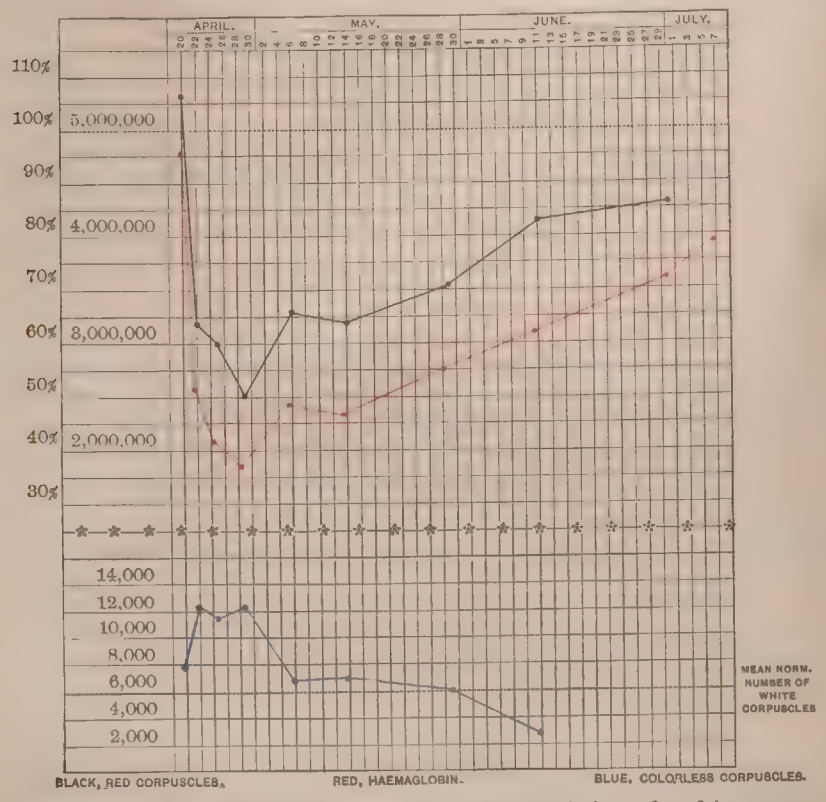

CHART XVII.—Illustrates the rapidity with which anæmia is produced in purpura hæmorrhagica and the gradual recovery.*

hæmoglobin is restored more slowly than the corpuscles. The accompanying chart illustrates the rapid fall and gradual restitution in a case of severe purpura hæmorrhagica.

The microscopical characters of the blood after severe hæmorrhage may not be greatly changed. The red corpuscles show, usually, rather more marked differences in size than normally, while the average size may be a trifle reduced; there may be a moderate poikilocytosis. The corpuscles are paler than normally. Nucleated red corpuscles appear, almost always, soon after the hæmorrhage; they are, however, not numerous. These are small bodies of about the same size as a normal red corpuscle with a small,

* On September 27th the patient returned from the country, where she had spent the summer. The blood-count was then: Red corpuscles, 5,350,000; white corpuscles, 5,500; hæmoglobin, 94 per cent.

round, deeply staining nucleus. Free nuclei may be found. The colorless corpuscles are, at first, increased in number. There is a moderate leucocytosis, the differential count showing an increase in the multinuclear neutrophiles with a diminution in the small mononuclear elements. During recovery the leucocytosis diminishes.

The reduction in hæmoglobin is always proportionately greater than that in the corpuscles.

In some instances a rapidly fatal anæmia may follow a single severe hæmorrhage, or repeated small hæmorrhages as in purpura. Here the appearances of the red corpuscles are much the same, except in the total absence of nucleated red corpuscles.

The leucocytes in these cases are usually reduced in number; the polynuclear elements are present in a relatively diminished proportion, while the small mononuclear forms are numerous. The autopsy, in these cases, reveals usually a total absence of any regenerative activity on the part of the bone-marrow.

(2) Anæmia is frequently produced by long-continued drain on the albuminous materials of the blood, as in chronic suppuration and Bright's disease. Prolonged lactation acts in the same way. Rapidly growing tumors may cause a profound anæmia, as in gastric cancer. The characters of the blood here may be much the same as in the acute cases. Usually, though, the poikilocytosis is much more marked; in severe cases it may be excessive. The presence, however, of the very large corpuscles, such as one sees in pernicious anæmia, is not noted, the average size appearing to be rather smaller than normal.

Nucleated red corpuscles are usually scanty. In long-continued chronic secondary anæmias occasional larger nucleated red corpuscles may be seen, bodies with larger palely staining nuclei; in some of these cells karyokinetic figures occur. Nucleated red corpuscles with fragmentary nuclei may also be seen.

The leucocytes may be increased in number, though in some severe chronic cases there may be a diminution.

(3) *Anæmia from Inanition.*—This may be brought about by defective food supply, or by conditions which interfere with the proper reception and preparation of the food, as in cancer of the œsophagus and chronic dyspepsia. The reduction of the blood mass may be extreme, but the plasma suffers proportionately more than the corpuscles, which, even in the wasting of cancer of the œsophagus, may not be reduced more than one half or three fourths. In some instances the reduction in the plasma may be so great that the corpuscles show an apparent increase.

(4) *Toxic anæmia,* induced by the action of certain poisons on the blood, such as lead, mercury, and arsenic, among inorganic substances, and the virus of syphilis and malaria among organic poisons. They act either by directly destroying the red blood-corpuscles, as in malaria, or by increasing the rate of ordinary consumption. The anæmia of pyrexia may in part be due to a toxic action, but is also caused in part by the disturbance of digestion and interference with the function of the blood-making organs.

Primary or Essential Anæmia.

1. Chlorosis.

Definition.—An anæmia of unknown cause, occurring in young girls, characterized by a marked relative diminution of the hæmoglobin.

Etiology.—It is a disease of girls, more often of blondes than of brunettes. It is doubtful if males are ever affected. I have never seen true chlorosis in a boy. The age of onset is between the fourteenth and seventeenth years; under the age of twelve cases are rare. Recurrences, which are common, may extend into the third decade. Of the essential cause of the disease we know nothing. There exists a lowered energy in the blood-making organs, associated in some obscure way with the evolution of the sexual apparatus in women. Hereditary influences, particularly chlorosis and tuberculosis, play a part in some cases. Sometimes, as Virchow pointed out, the condition exists with a defective development (hypoplasia) of the circulatory and generative organs.

The disease is most common among the ill-fed, overworked girls of large towns, who are confined all day in close, badly lighted rooms, or have to do much stair-climbing. Cases are frequent, however, under the most favorable conditions of life. Lack of proper exercise and of fresh air, and the use of improper food are important factors. Emotional and nervous disturbances may be prominent—so prominent that certain writers have regarded the disease as a neurosis. De Sauvages speaks of a *chlorose par amour*. Newly arrived Irish girls were very prone to the disease in Montreal. The "corset and chlorosis" expresses O. Rosenbach's opinion. Menstrual disturbances are not uncommon, but are probably a sequence, not a cause, of chlorosis. Sir Andrew Clark believed that constipation plays an important *rôle*, and that the condition is in reality a *copræmia* due to the absorption of poisons—leucomaines and ptomaines—from the large bowel, a view which always appeared to me baseless, considering the great frequency of the condition in women.

Symptoms.—(*a*) **General.**—The symptoms of chlorosis are those of anæmia. The subcutaneous fat is well retained or even increased in amount. The complexion is peculiar; neither the blanched aspect of hæmorrhage nor the muddy pallor of grave anæmia, but a curious yellow-green tinge, which has given to the disease its name, and its popular designation, the green sickness. Occasionally the skin shows areas of pigmentation, particularly about the joints. In cases of moderate grade the color may be deceptive, as the cheeks have a reddish tint, particularly on exertion (chlorosis rubra). The subjects complain of breathlessness and palpitation, and there may be a tendency to fainting—symptoms which often lead to the suspicion of heart or lung disease. Puffiness of the face and swelling of the ankles may suggest nephritis. The disposition often changes, and the girl becomes low-spirited and irritable. The eyes have a peculiar brilliancy and the sclerotics are of a bluish color.

(*b*) **Special Features.**—*Blood.*—The drop as expressed looks pale. Johann Duncan, in 1867, first called attention to the fact that the essen-

anaemia of a chlorotic type is not very infrequently seen in males. There are individuals who for years are below par in colour but who are active and vigorous, and whose red blood cells are up to or above normal, while the haemoglobin may not be more than 70%.

tial feature was not a great reduction in the number of the corpuscles, but a quantitative change in the hæmoglobin. The corpuscles themselves look pale. In 63 consecutive cases examined at my clinic by Thayer, the average number per cubic millimetre of the red blood-corpuscles was 4,096,544, or over 80 per cent, whereas the percentage of hæmoglobin for the total number was 42.3 per cent. The accompanying chart illustrates well these striking differences. There may, however, be well-marked actual anæmia. The lowest blood-count in the series of cases referred to above was 1,932,000. There may be all the physical characteristics and symptoms of a profound anæmia with the number of the blood-corpuscles nearly at the normal

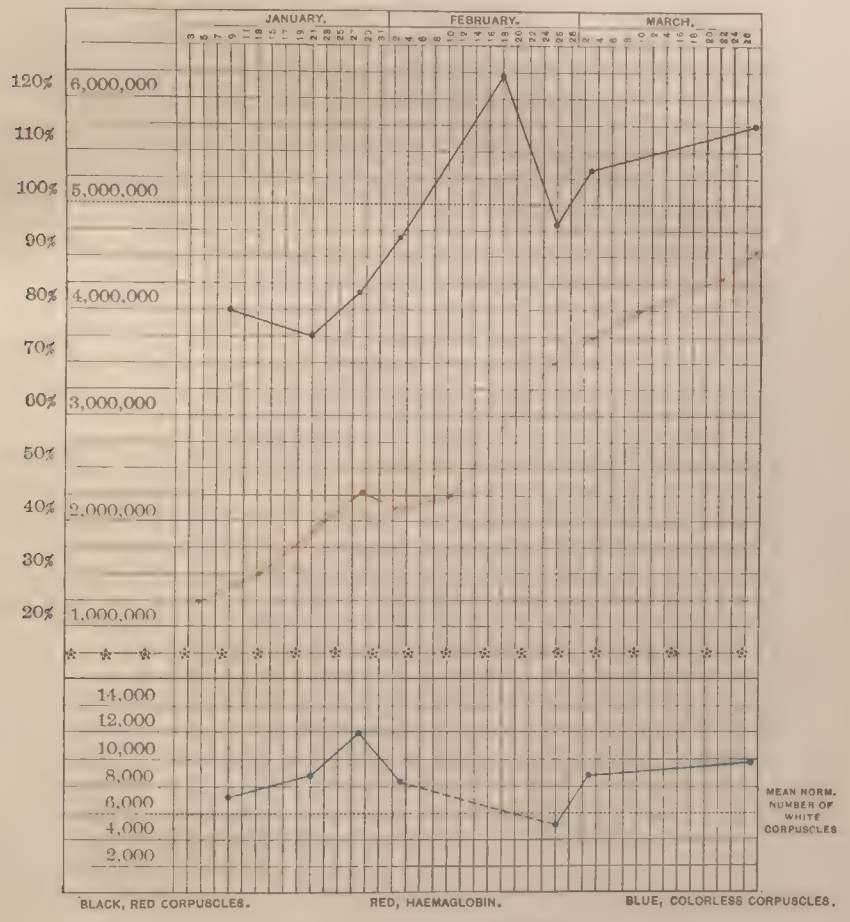

CHART XVIII.—Chlorosis.

standard. Thus in one instance the globular richness was over 85 per cent, with the hæmoglobin about 35. No other form of anæmia presents this feature, at least with the same constancy and in the same degree. The importance of the reduction in the hæmoglobin depends upon the fact that

it is the iron-containing elements of the blood with which in respiration the oxygen enters into combination. This marked diminution in the iron has also been determined by chemical analysis of the blood. The microscopical characteristics of the blood are as follows: In severe cases the corpuscles may be extremely irregular in size and shape—poikilocytosis, which may occasionally be as marked as in some cases of pernicious anæmia. The large forms of red blood-cells are not as common, and the average size is stated to be below normal. The color of the corpuscles is noticeably pale and the deficiency may be seen either in individual corpuscles or in the blood mixture prepared for counting. Nucleated red corpuscles (normoblasts) are not very uncommon, and may vary greatly in numbers in the same case at different periods. The leucocytes may show a slight increase; the average in the 63 cases above referred to was 8,467 per cubic millimetre.

(*c*) **Gastro-intestinal Symptoms.**—The appetite is capricious, and patients often have a longing for unusual articles, particularly acids. In some instances they eat all sorts of indigestible things, such as chalk or even earth. Superacidity of the gastric juice is commonly associated with chlorosis. In 19 out of 21 cases in Riegel's clinic this condition was found to exist. In the other two instances the acidity was normal or a trifle increased. Distress after eating and even cardialgic attacks may be associated with it. Constipation is a common symptom, and, as already mentioned, has been regarded as an important element in causing the disease. A majority of chlorotic girls who wear corsets have gastroptosis, and on inflation the stomach will be found vertically placed; sometimes the organ is very much dilated. The motor power is usually well retained. Enteroptosis with palpable right kidney is not uncommon.

(*d*) **Circulatory Symptoms.**—Palpitation of the heart occurs on exertion, and may be the most distressing symptom of which the patient complains. Percussion may show slight increase in the transverse dulness. A systolic murmur is heard at the apex or at the base; more commonly at the latter, but in extreme cases at both. A diastolic murmur is rarely heard. The systolic murmur is usually loudest in the second left intercostal space, where there is sometimes a distinct pulsation. The exact mode of production is still in dispute. Balfour holds that it is produced at the mitral orifice by relative insufficiency of the valves in the dilated condition of the ventricle. On the right side of the neck over the jugular vein a continuous murmur is heard, the *bruit de diable*, or humming-top murmur.

The pulse is usually full and soft. Pulsation in the peripheral veins is sometimes seen. There is a tendency to thrombosis in the veins; most commonly in the femoral, but in other instances in the longitudinal sinus; or the thrombosis may be multiple. Except in the sinuses, the condition is rarely serious. Tuckwell has reported an instance in which there was embolism of the right axillary artery with the loss of a thumb and part of the fingers. Brayton Ball has recently called attention to the importance of this feature of chlorosis.

As in all forms of essential anæmia, fever is not uncommon. Especial attention has of late been directed to this by French writers. Chlorotic

patients suffer frequently from headache and neuralgia, which may be paroxysmal. The hands and feet are often cold. Dermatographia is common. Hysterical manifestations are not infrequent. Menstrual disturbances are very common—amenorrhœa or dysmenorrhœa. With the improvement in the blood condition this function is usually restored.

Diagnosis.—The green sickness, as it is sometimes called, is in many instances recognized at a glance. The well-nourished condition of the girl, the peculiar complexion, which is most marked in brunettes, and the white or bluish sclerotics are very characteristic. A special danger exists in mistaking the apparent anæmia of the early stage of pulmonary tuberculosis for chlorosis. Mistakes of this sort may often be avoided by the very simple test furnished by allowing a drop of blood to fall on a white towel or a piece of blotting paper—a deficiency in hæmoglobin is readily appreciated. The palpitation of the heart and shortness of breath frequently suggest heart-disease, and the œdema of the feet and general pallor cause the cases to be mistaken for Bright's disease. In the great majority of cases the characters of the blood readily separate chlorosis from other forms of anæmia.

2. Idiopathic or Progressive Pernicious Anæmia.

The disease was first clearly described by Addison, who called it idiopathic anæmia. Channing and Gusserow described the cases occurring post partum, but to Biermer we owe a revival of interest in the subject.

Etiology.—The existence of a separate disease worthy of the term progressive pernicious anæmia has been doubted, but there are unquestionably cases in which, as Addison says, there exist none of the usual causes or concomitants of anæmia. Clinically there are several different groups which present the characters of a progressive and pernicious anæmia and are etiologically different. Thus, a fatal anæmia may be due to the presence of parasites, or may follow hæmorrhage, or be associated with chronic atrophy of the stomach; but when we have excluded all these causes there remains a group which, in the words of Addison, is characterized by a "general anæmia occurring without any discoverable cause whatever, cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease."

Idiopathic anæmia is widely distributed. It is of frequent occurrence in the Swiss cantons, and it is not uncommon in this country. It affects middle-aged persons, but instances in children have been described. Griffith mentions about 10 cases occurring under twelve years of age. The youngest patient I have seen was a girl of twenty. Males are more frequently affected than females. Of my 27 cases, 10 were females and 17 were males. Of 110 cases collected by Coupland, 56 were in men and 54 in women. Sinkler and Eshner give 3 cases in one family, the father and two girls; the father had symptoms of posterior sclerosis.

With the following conditions may be associated a profound anæmia not to be distinguished clinically from Addison's idiopathic form:

(*a*) *Pregnancy and Parturition.*—The symptoms may develop during pregnancy, as in 19 of 29 cases of this group in Eichhorst's table. More commonly, in my experience, the condition has been post partum; thus, of my 27 cases, 5 followed delivery.

(*b*) *Atrophy of the Stomach.*—This condition, early recognized by Flint and Fenwick, may certainly cause a progressive pernicious anæmia. By modern methods it may now be possible to exclude this extreme gastric atrophy.

(*c*) *Parasites.*—The most severe form may be due to the presence of parasites, and the accounts of cases depending upon the anchylostoma and the bothriocephalus describe a progressive and often pernicious anæmia.

After the exclusion of these forms there remains a large proportion, numbering 18 cases in my series, which correspond to Addison's description. The etiology of these cases is still dark. The researches of Quincke and his student Peters showed that there was an enormous increase in the iron in the liver, and they suggested that the affection was probably due to increased hæmolysis. This has been strongly supported by the extensive observations of Hunter, who has also shown that the urine excreted is darker in color and contains pathological urobilin. The lemon tint of the skin or the actual jaundice is attributed, on this view, to an overproduction. To explain the hæmolysis, it has been thought that in the condition of faulty gastro-intestinal digestion, which is so commonly associated with these cases, poisonous materials are developed, which when absorbed cause destruction of the corpuscles. Certainly the evidence for hæmolysis is very strong, but we are still far away from a full knowledge of the conditions under which it is produced.

Stockman suggests that repeated small capillary hæmorrhages—chiefly internal—play an important *rôle* in the causation of the disease, which also explains, he holds, the existence of a great excess of iron in the liver.

On the other hand, F. P. Henry, Stephen Mackenzie, Rindfleisch, and other authorities incline to the belief that the essence of the disease is in defective hæmogenesis, in consequence of which the red blood-corpuscles are abnormally vulnerable. A point noted by Copeman, that the hæmoglobin crystallizes from the blood-corpuscles with great readiness, can scarcely be regarded as favoring the view of imperfect hæmogenesis, since this is a feature specially characteristic of the blood of the young.

Morbid Anatomy.—The body is rarely emaciated. A lemon tint of the skin is present in a majority of the cases. The muscles often are intensely red in color, like horse-flesh, while the fat is light yellow. Hæmorrhages are common on the skin and serous surfaces. The heart is usually large, flabby, and empty. In one instance I obtained only 2 drachms of blood from the right heart, and between 3 and 4 from the left. The muscle substance of the heart is intensely fatty, and of a pale, light-yellow color. In no affection do we see more extreme fatty degeneration. The lungs show no special changes. The stomach in many instances is normal, but in some cases of fatal anæmia the mucosa has been extensively atrophied. In the case described by Henry and myself the mucous membrane had a smooth, cuticular appearance, and there was complete atrophy of

the secreting tubules. The liver may be enlarged and fatty. In most of my autopsies it was normal in size, but usually fatty. The iron is in excess, a striking contrast to the condition in cases of secondary anæmia. It is deposited in the outer and middle zones of the lobules, and in two specimens, which I examined, seemed to have such a distribution that the bile capillaries were distinctly outlined. This, Hunter states, is a special and characteristic lesion, possibly peculiar to pernicious anæmia. A. J. Scott examined for me the livers in 45 consecutive autopsies without finding (except in pernicious anæmia) this special distribution of pigment.

The spleen shows no important changes. In one of Palmer Howard's cases the organ weighed only 1 ounce and 5 drachms. The iron pigment is usually in excess. The lymph-glands may be of a deep red color. The amount of iron pigment is increased in the kidneys, chiefly in the convoluted tubules. The bone marrow, as pointed out by H. C. Wood, is usually red, lymphoid in character, showing great numbers of nucleated red corpuscles, especially the larger forms called by Ehrlich gigantoblasts. Changes in the ganglion cells of the sympathetic have been reported on several occasions. Lichtheim has found sclerosis in the posterior columns of the cord. Burr described a series of cases. The subject is referred to again under diseases of the spinal cord (University Med. Magazine, 1895).

Symptoms.—The patient may have been in previous good health, but in many cases there is a history of gastro-intestinal disturbance, mental shock, or worry. The description given by Addison presents the chief features of the disease in a masterly way. "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

The Blood.—The corpuscles may fall to one fifth or less of the normal number. They may sink to 500,000 per cubic millimetre, and in a case of Quincke's the number was reduced to 143,000 per cubic millimetre. The hæmoglobin is relatively increased, so that the individual globular richness is plus, a condition exactly the opposite to that which occurs in

chlorosis and the secondary anæmia, in which the corpuscular richness in coloring matter is minus. The relative increase in the hæmoglobin is probably associated with the average increase in the size of the red blood-corpuscles. The accompanying chart illustrates these points. Microscopically the red blood-corpuscles present a great variation in size, and there can be seen large giant forms, megalocytes, which are often ovoid in form,

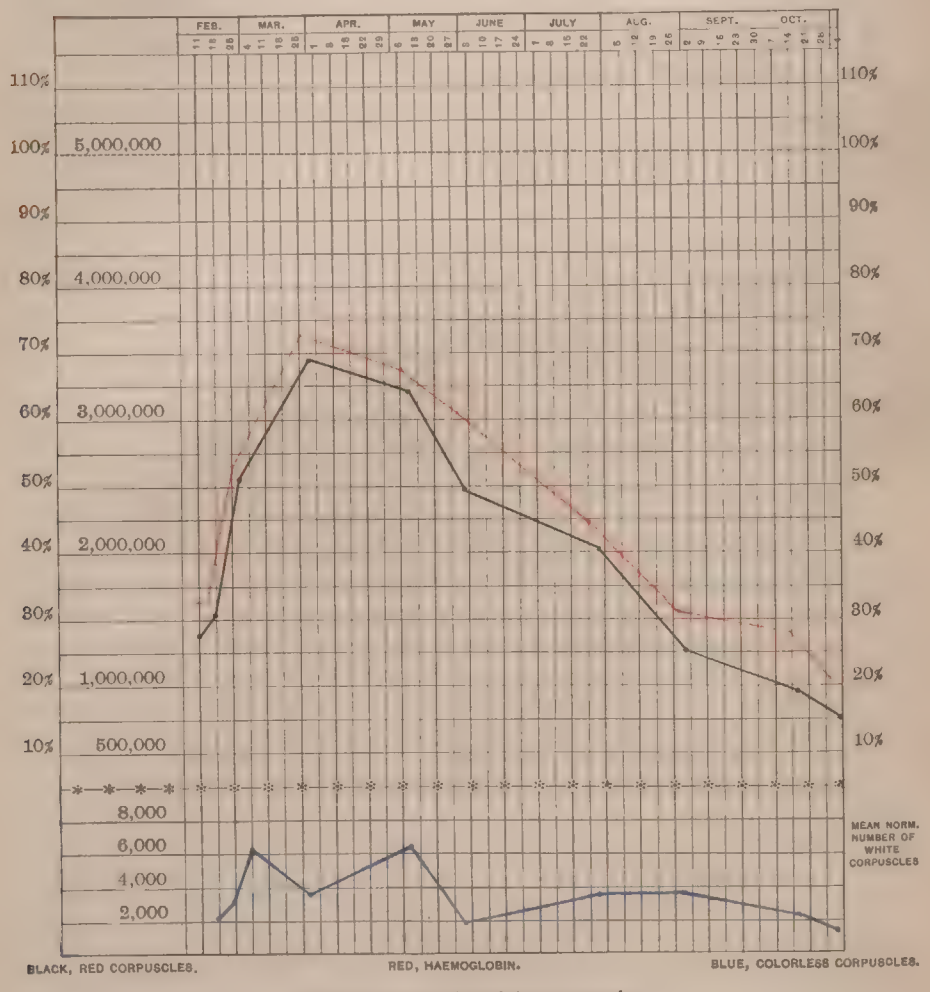

CHART XIX.—Pernicious anæmia.

measuring 8, 11, or even 15 μ in diameter—a circumstance which Henry regards as indicating a reversion to a lower type. Laache thinks these pathognomonic, and they certainly form a constant feature. There are also small round cells, microcytes, from 2 to 6 μ in diameter, and of a deep red color. The corpuscles show a remarkable irregularity in form; they are elongated and rodlike or pyriform; one end of a corpuscle may

retain its shape while the other is narrow and extended. To this condition of irregularity Quincke gave the name poikilocytosis.

Nucleated red blood-corpuscles are almost always present, as pointed out by Ehrlich. Besides the ordinary form, which is of the same size as the common corpuscle and which has a small, deeply stained nucleus (normoblasts), there are very large forms with palely staining nuclei (gigantoblasts), which resemble somewhat the larger megalocytes. Ehrlich regards the presence of these as almost distinctive of progressive pernicious anæmia. Though these large forms are most characteristic, occasionally forms closely similar to them may be found in the graver secondary anæmias—e. g., bothriocephalus anæmia, anchylostomiasis—and in leukæmia. Karyokinetic figures may be seen in these bodies. Red corpuscles with fragmenting nuclei are common in pernicious anæmia. The leucocytes are generally normal or diminished in number; and in the graver cases a marked relative increase in the small mononuclear forms, with a diminution in the polynuclear leucocytes, is often noted. The blood-plates are either absent or very scanty.

The cardio-vascular symptoms are important and are noted in the description given above. Hæmic murmurs are constantly present. The larger arteries pulsate visibly and the throbbing in them may be distressing to the patient. The pulse is full and frequently suggests the water-hammer beat of aortic insufficiency. The capillary pulse is frequently to be seen. The superficial veins are often prominent, and in 2 cases I have seen well-marked pulsation in them. Hæmorrhages may occur, either in the skin or from the mucous surfaces. Retinal hæmorrhages are common. There are rarely symptoms in the respiratory organs.

Gastro-intestinal symptoms, such as dyspepsia, nausea, and vomiting, may be present throughout the disease. Diarrhœa is not infrequent. The urine is usually of a low specific gravity and sometimes pale, but in other instances it is of a deep sherry color, shown by Hunter and Mott to be due to great excess of urobilin. Fever is a variable symptom. For weeks at a time the temperature may be normal, and then irregular pyrexia may develop. Nervous symptoms may occur, numbness and tingling, and occasionally symptoms resembling those of tabes. Lepine reports a case of extensive paralysis.

Diagnosis.—From chlorosis the disease is readily distinguished. I have not seen a case in which the two diseases could have been confounded. Several points in the blood examination are of especial importance, namely, the relative increase in the hæmoglobin and the presence of megalocytes and of the large forms of nucleated red blood-corpuscles, the gigantoblasts of Ehrlich. Poikilocytosis may occur in any severe anæmia. The separation of the different clinical forms above referred to can usually be made. The profound secondary anæmia of cancer of the stomach may sometimes be puzzling, but the skin is rarely, if ever, lemon-tinted, and the blood has the characteristics of a secondary, not a primary, anæmia.

Prognosis.—In the true Addisonian cases the outlook is bad, though of late years on the arsenic treatment the proportion of recovery has increased. My personal experience of progressive pernicious anæmia to Janu-

ary, 1895, was as follows: Of 27 cases, 4 were then under observation, 2 of these having recovered with arsenic. Of the remaining 23, 4 of the 5 post-partum cases recovered, and when I left Montreal 3 of these cases had remained in good health for several years. Of the remaining 18 cases 2 were lost sight of; 1 had improved very much. The remaining 16 were dead. Six of these fatal cases recovered from the first attack; one had an interval of nearly three years, and another nearly two years, before the return. One patient in hospital in 1890 recovered completely, and died in 1896 of cancer of the stomach. In Pye-Smith's article in the Guy's Hospital Reports, he mentions 20 cases of recovery. Hale White, in a recent article, states that one of these cases, treated with arsenic in 1880, remained alive and well January, 1891. One of my patients made an apparently complete recovery and resumed active business and political duties. So characteristic are recurrences in this affection that Stephen Mackenzie, in his lectures, considered them under a separate heading of relapsing pernicious anæmia. The examination of the blood may give us some help. The presence of numerous normoblasts appears in some instances to be indicative of an active regeneration in the marrow. Cases in which a majority of the nucleated red corpuscles are gigantoblasts are generally more malignant. A marked relative increase in the small mononuclear leucocytes appears to be also an unfavorable sign.

Treatment of Anæmia.—*Secondary Anæmia.*—The traumatic cases do best, and with plenty of good food and fresh air the blood is readily restored. The extraordinary rapidity with which the normal percentage of red blood-corpuscles is reached without any medication whatever is an important lesson. The cause of the hæmorrhage should be sought and the necessary indications met. The large group depending on the drain on the albuminous materials of the blood, as in Bright's disease, suppuration, and fever, is difficult to treat successfully, and so long as the cause keeps up it is impossible to restore the normal blood condition. The anæmia of inanition requires plenty of nourishing food. When dependent on organic changes in the gastro-intestinal mucosa not much can be expected from either food or medicine. In the toxic cases due to mercury and lead, the poison must be eliminated and a nutritious diet given with full doses of iron. In a great majority of these cases there is deficient blood formation, and the indications are briefly three: plenty of food, an open-air life, and iron. As a rule it makes but little difference what form of the drug is administered.

The treatment of *chlorosis* affords one of the most brilliant instances—of which we have but three or four—of the specific action of a remedy. Apart from the action of quinine in malarial fever, and of mercury and iodide of potassium in syphilis, there is no other drug the beneficial effects of which we can trace with the accuracy of a scientific experiment. It is a minor matter *how* the iron cures chlorosis. In a week we give to a case as much iron as is contained in the entire blood, as even in the worst case of chlorosis there is rarely more than a deficit of 2 grammes of this metal. Iron is present in the fæces of chlorotic patients before they are placed upon any treatment, so that the disease does not result from any

deficiency of available iron in the food. Bunge believes that it is the sulphur which interferes with the digestion and assimilation of this natural iron. The sulphides are produced in the process of fermentation and decomposition in the fæces, and interfere with the assimilation of the normal iron contained in the food. By the administration of an inorganic preparation of iron, with which these sulphides unite, the natural organic combinations in the food are spared. In studying a number of charts of chlorosis, it is seen that there is an increase in the red blood-corpuscles under the influence of the iron, and in some instances the globular richness rises above normal. The increase in the hæmoglobin is slower and the maximum percentage may not be reached for a long time. I have for years in the treatment of chlorosis used with the greatest success Blaud's pills, made and given according to the formula in Niemeyer's text-book, in which each pill contains 2 grains of the sulphate of iron. During the first week one pill is given three times a day; in the second week, two pills; in the third week, three pills, three times a day. This dose should be continued for four or five weeks at least before reduction. An important feature in the treatment of chlorosis is to persist in the use of the iron for at least three months, and, if necessary, subsequently to resume it in smaller doses, as recurrences are so common. The diet should consist of good, easily digested food. Special care should be directed to the bowels, and if constipation is present a saline purge should be given each morning. Such stress does Sir Andrew Clark lay on the importance of constipation in chlorosis, that he states that if limited to the choice of one drug in the treatment of the disease he would choose a purgative. The good influence of alkaline waters in association with the treatment by iron has been noted by von Jaksch. In many instances the dyspeptic symptoms may be relieved by alkalies and a treatment directed toward a moderate superacidity. Dilute hydrochloric acid, manganese, phosphorus, and oxygen have been recommended.

Treatment of Pernicious Anæmia.—Since the introduction by Byrom Bramwell of arsenic in this affection a large number of cases have been temporarily, a few permanently, cured by it. It should be given as Fowler's solution in increasing doses. It is usually well borne, and patients, as a rule, take up to 20 minims three times a day without any disturbance. I usually begin with 3 minims and increase to 5 at the end of the first week, to 10 at the end of the second week, to 15 at the end of the third week, and, if necessary, go up to 20 or 25. In a case in which the recovery persisted for nearly three years the dose was gradually increased to 30 minims. These patients seem to stand the arsenic extremely well. It is sometimes better borne as arsenious acid in pill form. Vomiting and diarrhœa are rare; occasionally puffiness of the face is produced, and in some cases pigmentation of the skin.

Rest in bed and a light but nutritious diet (giving the food in small amounts and at fixed intervals) are the first indications. I always prefer to begin the treatment of a case of pernicious anæmia, whatever the grade may be, with rest in bed as one of the essential elements. The beneficial effect of massage has been shown by J. K. Mitchell. I have abandoned

the use of rectal injections of dried blood. Iron rarely acts well in this form, but in a case in which the arsenic disagrees it may be tried. Bone marrow has been recommended. It is best given as a glycerin extract. I have not seen any benefit follow its administration. Inhalations of oxygen may be tried.

II. LEUKÆMIA.

Definition.—An affection characterized by persistent increase in the white blood-corpuscles, associated with changes, either alone or together, in the spleen, lymphatic glands, or bone marrow.

The disease was described almost simultaneously by Virchow and by Bennett, who gave to it the name leucocythæmia. It is ordinarily seen in two main types, though combinations and variations may occur:

(1) Spleno-medullary leukæmia, in which the changes are especially localized in the spleen and the bone marrow, while the blood shows a great increase in elements which are derived especially from the latter tissue, a condition which Müller has termed "myelæmia." Ehrlich prefers to call this type of the disease "myelogenous leukæmia," believing the part played by the spleen in the process to be purely passive.

(2) Lymphatic leukæmia, in which the changes are chiefly localized in the lymphatic apparatus, the blood showing an especial increase in those elements derived from the lymph-glands.

Etiology.—We know nothing of the conditions under which the disease develops. It is not uncommon on this continent. Of 26 cases of which I have notes, to January, 1895, 11 occurred in Montreal, 2 in Philadelphia, and 13 in hospital and private work in Baltimore. It does not seem more frequent in the southern parts of the country.

The disease is most common in the middle period of life. The youngest of my patients was a child of eight months, and cases are on record of the disease as early as the eighth or tenth week. It may occur as late as the seventieth year. Males are more prone to the affection than females. Of my cases, 17 were in males and 9 in females. Birch-Hirschfeld states that of 200 cases collected from the literature, 135 were males and 65 females.

A tendency to hæmorrhage has been noted in many cases, and some of the patients have suffered repeatedly from nose-bleeding. In women the disease is most common at the climacteric. There are instances in which it has developed during pregnancy. The case described by J. Chalmers Cameron, of Montreal, is in this respect remarkable, as the patient passed through three pregnancies, bearing on each occasion non-leukæmic children. The case is interesting, too, as showing the hereditary character of the affection, as the grandmother and mother, as well as a brother, suffered from symptoms strongly suggestive of leukæmia. One of the patient's children had leukæmia before the mother showed any signs, and a second died of the disease. At the last report this patient had gradually recovered from the third confinement, and the red blood-corpuscles had risen to 4,000,000 per cubic millimetre, and the ratio of white to red was 1

to 200. Sänger has reported a case in which a healthy mother bore a leukæmic child.

Malaria is believed by some to be an etiological factor. Of 150 cases analyzed by Gowers, there was a history of malaria in 30; in my series there was a history in at least 9. Syphilis appears in some cases to have been closely associated with leukæmia. The disease has followed injury or a blow.

The lower animals are subject to the affection, and cases have been described in horses, dogs, oxen, cats, swine, and mice.

Morbid Anatomy.—The wasting may be extreme, and dropsy is sometimes present. There is in many cases a remarkable condition of polyæmia; the heart and veins are distended with large blood-clots. In Case XI of my series the weight of blood in the heart chambers alone was 620 grammes. There may be remarkable distention of the portal, cerebral, pulmonary, and subcutaneous veins. The blood is usually clotted, and the enormous increase in the leucocytes gives a pus-like appearance to the coagula, so that it has happened more than once, as in Virchow's memorable case, that on opening the right auricle the observer at first thought he had cut into an abscess. The coagula have a peculiar greenish color, somewhat like the fat of a turtle. The alkalinity of the blood is diminished. The fibrin is increased. The character of the corpuscles will be described under the symptoms. Charcot's octohedral crystals may separate from the blood after death. The specific gravity of the blood is somewhat lowered. There may be pericardial ecchymoses.

In the spleno-medullary form the spleen is greatly enlarged. Strong adhesions may unite it to the abdominal wall, the diaphragm, or the stomach. The capsule may be thickened. The vessels at the hilus are enlarged; the weight may range from 2 to 18 pounds. The organ is in a condition of chronic hyperplasia. It cuts with resistance, has a uniformly reddish-brown color, and the Malpighian bodies are invisible. Grayish-white, circumscribed, lymphoid tumors may occur throughout the organ, contrasting strongly with the reddish-brown matrix. In the early stage the swollen spleen pulp is softer, and it is stated that rupture has occurred from the intense hyperæmia.

In association with these changes in the spleen, the bone marrow is involved, the lieno-medullary form of the Germans. The essential change, indeed, in the disease appears to be the extraordinary hyperplasia of the red marrow, and the appearance of an hyperplastic cellular tissue in regions where in the adults the marrow is fatty. Instead of a fatty tissue, the medulla of the long bones may resemble the consistent matter which forms the core of an abscess, or it may be dark brown in color. In Ponfick's case there were hæmorrhagic infarctions. There may be much expansion of the shell of bone, and localized swellings which are tender and may even yield to firm pressure. Histologically, there are found in the medulla large numbers of nucleated red corpuscles in all stages of development, numerous cells with eosinophilic granules, both small polynuclear forms and large almost giant mononuclear elements. There are also many large cells with single large nuclei and neutrophilic granules—the cellules

medullaires of Cornil—the *myelocytes* which are found in the blood. Great numbers of polynuclear leucocytes are also present, as well as a certain number of small mononuclear elements.

In the lymphatic forms of the disease there is a general lymphatic enlargement, which is usually associated with a certain amount of enlargement of the spleen. In only one of my cases was the splenic enlargement notable. In the cases of lymphatic leukæmia the cervical, axillary, mesenteric, and inguinal groups may be much enlarged, but the glands are usually soft, isolated, and movable. They may vary considerably in size during the course of the disease. The tonsils and the lymph follicles of the tongue, pharynx, and mouth may be enlarged. Numerous mitoses may be found in the small cells of the lymphatic tissue.

In some instances there are leukæmic enlargements in the solitary and agminated glands of Peyer. In a case of Willcocks' there were growths on the surface of the stomach and gastro-splenic omentum. The thymus is rarely involved, though it has been enlarged in some of the cases of acute lymphatic leukæmia. The bone marrow in these cases may be replaced by a lymphoid tissue. Nucleated red corpuscles and the normal granular marrow elements may be greatly reduced in number.

The liver may be enlarged, and in a case described by Welch it weighed over 13 pounds. The enlargement is usually due to a diffuse leukæmic infiltration. The columns of liver cells are widely separated by leucocytes, which are partly within and partly outside the lobular capillaries. There may be definite leukæmic growths.

There are rarely changes of importance in the lungs. The kidneys are often enlarged and pale, the capillaries may be distended with leucocytes, and leukæmic tumors may occur. The skin may be involved, as in a case described by Kaposi.

Leukæmic tumors in the organs are not common. They were present in only 1 of the 12 autopsies in my series. In 159 cases collected by Gowers there were only 13 instances of leukæmic nodules in the liver and 10 in the kidneys. These new growths probably develop from leucocytes which leave the capillaries. Bizzozero has shown that the cells which compose them are in active fission.

Symptoms.—The onset is insidious, and, as a rule, the patient seeks advice for progressive enlargement of the abdomen and shortness of breath, or for the enlarged glands or the pallor, palpitation, and other symptoms of anæmia. Bleeding at the nose is common. Gastro-intestinal symptoms may precede the onset. Occasionally the first symptoms are of a very serious nature. In one of the cases of my series the boy played lacrosse two days before the onset of the final hæmatemesis; and in another case a girl, who had, it was supposed, only a slight chlorosis, died of fatal hæmorrhage from the stomach before any suspicion had been aroused as to the true condition.

Anæmia is not a necessary accompaniment of all stages of the disease; the subjects may look very healthy and well.

As has been stated, the disease is most commonly seen in two main types, though combinations may occur.

Lantanie kinnors many ophear vereelt ll.
vermijdelijk (alten)

hæmorrhage [illegible] pleura or
peritoneum

Sudden death [illegible] in the
post case in Trans. Path. Soc. Vol. 14. 1863.

Skin lesions.

H. Dercteloftheu Haven - (Yale Med. Journ. Jan. 1899) describes a very interesting case with multiple, small, round, hard spots in the skin. It was thought at first to be multiple sarcomata, but the blood examination showed it to be leukaemia, and examination of the abdomen showed an enormous spleen.

↑ Facial paralysis has been noted.

↑ Features like Menieres disease may come on quite suddenly, due to leukaemic infiltration or haemorrhage into the semi-circular canal.
(H. Parker Heber, Med. Chir. Trans. Vol. 83)

(1) Spleno-medullary Leukæmia.

This is much the commonest type of the disease. The gradual increase in the volume of the spleen is the most prominent symptom in a majority of the cases. Pain and tenderness are common, though the progressive enlargement may be painless. A creaking fremitus may be felt on palpation. The enlarged organ extends downward to the right, and may be felt just at the costal edge, or when large it may extend as far over as the navel. In many cases it occupies fully one half of the abdomen, reaching to the pubes below and extending beyond the middle line. As a rule, the edge, in some the notch or notches, can be felt distinctly. Its size varies greatly from time to time. It may be perceptibly larger after meals. A hæmorrhage or free diarrhœa may reduce the size. The pressure of the enlarged organ may cause distress after eating; in one case it caused fatal obstruction of the bowels. A murmur may sometimes be heard over the spleen, and Gerhardt has described a pulsation in it.

The pulse is usually rapid, soft, compressible, but often full in volume. There are rarely any cardiac symptoms. The apex beat may be lifted an interspace by the enlarged spleen. Toward the close, as a consequence of the feeble circulation, œdema may occur in the feet or there may be general anasarca. Hæmorrhage is a common symptom and may be either late or early. There may be most extensive purpura. Epistaxis is the most frequent form. Hæmoptysis and hæmaturia are rare. Bleeding from the gums may be present. Hæmatemesis proved fatal in two of my cases, and in a third a large cerebral hæmorrhage rapidly killed. The leukæmic retinitis is a part of the hæmorrhagic manifestations.

Local gangrene may develop, with signs of intense infection and high fever. There are very few pulmonary symptoms. The shortness of breath is due, as a rule, to the anæmia. Toward the end there may be œdema of the lungs, or pneumonia may carry off the patient. The gastro-intestinal symptoms are rarely absent. Nausea and vomiting are early features in some cases. Diarrhœa may be very troublesome, even fatal. Intestinal hæmorrhage is not common. There may be a dysenteric process in the colon. Jaundice rarely occurs, though in one case of my series there were recurrent attacks. Ascites may be a prominent symptom, probably due to the presence of the splenic tumor. A leukæmic peritonitis also may be present, due to new growths in the membranes.

The nervous system is not often involved. Headache, dizziness, and fainting spells are due to anæmia. The patients are usually tranquil and resigned. Sudden coma may follow cerebral hæmorrhage.

The special senses are often affected. There is a peculiar retinitis, due chiefly to the extravasation of blood, but there may be aggregations of leucocytes, forming small leukæmic growths. Optic neuritis is rare. Deafness has frequently been observed; it may appear early and possibly is due to hæmorrhage.

The urine presents no constant changes. The uric acid excreted is always in excess, and possibly, as Salkowski suggests, stands in direct relation to the splenic tumor, or to the abundant leucocytes.

Priapism is a curious symptom which has been present in a large num-

ber of cases. It may, as in one of Edes' cases, be the first symptom. Peabody reports a case in which it persisted for six weeks. The cause is not known.

Slight fever is present in a majority of cases. Periods of pyrexia may alternate with prolonged intervals of freedom. The temperature may range from 102° to 103°.

Blood.—In all forms of the disease the diagnosis must be made by the examination of the blood, as it alone offers distinctive features.

The most striking change in the more common form, the lienomyelogenic, is the increase in the colorless corpuscles. The average normal number of white per cubic millimetre is estimated at about 6,000–7,000; thus the proportion of white to red is 1 to 500–1,000. In leukæmia the proportion may be 1 to 10, or 1 to 5, or may even reach 1 to 1. There are instances on record in which the number of leucocytes has exceeded that of the red corpuscles.

The character of the cells in splenic myelogenous leukæmia is as follows: The small mononuclear forms are little if at all increased; relatively they are greatly diminished. The eosinophiles are present in normal or increased relative proportion, so that there is a great total increase, and their presence is a striking feature in the stained blood-slide. The polynuclear neutrophiles may be in normal proportion; more frequently they are relatively diminished, and in the later stages they may form but a small proportion of the colorless elements. Marked differences in size between individual polynuclear leucocytes may be noted; the same is true of the eosinophiles. The most characteristic features of the blood in this form of leukæmia is the presence of cells which do not occur in normal blood. They appear to be derived from the marrow, and are called by Ehrlich *myelocytes*. They are considerably larger than the large mononuclear leucocytes, and are similar to them in appearance, but differ from them in the fact that the protoplasm is filled with the fine neutrophilic granules. Müller has recently found many large mononuclear elements with karyokinetic figures in leukæmic blood and in the marrow. These probably correspond to the myelocytes of Ehrlich as well as to the "cellules médullaires" of Cornil. Polynuclear cells with coarse basophilic granules, "Mastzellen," are always present in this form of leukæmia in considerable numbers. The granules do not stain in Ehrlich's triacid mixture, and the cells may be recognized as polynuclear non-granular elements. These cells, which form only about 0.28 per cent of the leucocytes of normal blood, may be even more numerous than the eosinophiles.

Nucleated red blood-corpuscles are present in considerable numbers. These are usually "normoblasts," but cells with larger paler nuclei, some showing evidences of mitosis, may be seen. Red cells with fragmented nuclei are common, while true gigantoblasts may be found. There is, as a rule, only a moderate reduction in the number of red blood-corpuscles; the number is rarely under 2,000,000 per cubic millimetre. The hæmoglobin is usually reduced in a somewhat greater proportion. The accompanying blood chart is from a case of leukæmia with an enormously enlarged spleen. Among other points about leukæmic blood may be men-

tioned the feebleness of the amœboid movement, as noted by Cafavy, which may be accounted for by the large number of mononuclear elements present, the polynuclear alone possessing this power. The blood-plates exist in variable numbers; they may be remarkably abundant. The fibrin network between the corpuscles is usually thick and dense. In blood-slides which

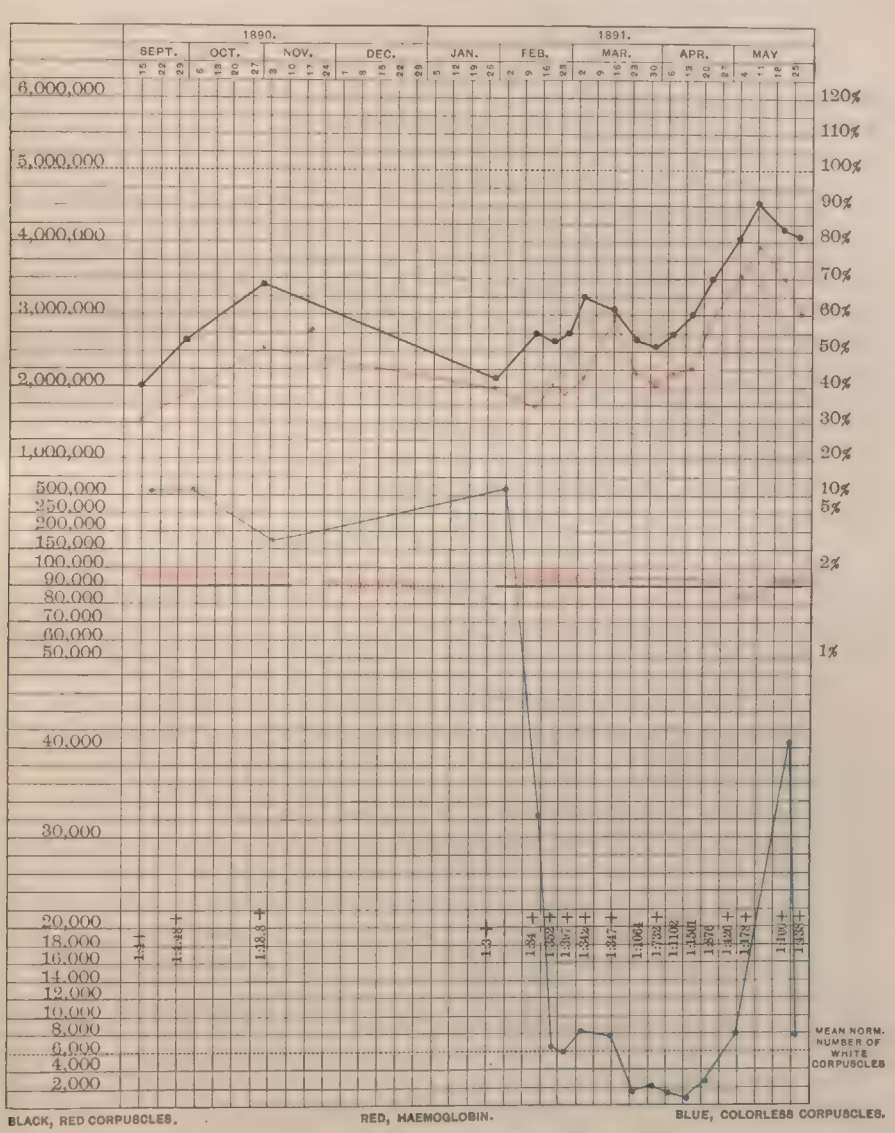

CHART XX.—Leukæmia.

are kept for a short time, Charcot's octohedral crystals separate, and in the blood of leukæmia the hæmoglobin shows a remarkable tendency to crystallize.

2. Lymphatic Leukæmia.

This form of leukæmia is rare. As mentioned, in but 4 of my series of 26 cases were the glands enlarged. The superficial groups are usually most involved, and even when affected it is rare to see such large bunches as in Hodgkin's disease. External lymph tumors are rare. Lymphatic leukæmia is often more rapid and fatal in its course, though chronic cases may occur. It is more common in young subjects.

The histological characters of the *blood* in lymphatic leukæmia differ materially from those in the spleno-medullary form. The increase in the colorless elements is never so great as in the preceding form; a proportion of 1 to 10 would be extreme. This increase takes place solely in the lymphocytes, all other forms of leucocytes being present in greatly diminished relative proportion. In one of my cases over 98 per cent of all the leucocytes were lymphocytes. In some cases, as Cabot has pointed out, this increase takes place largely in the smaller forms, while in others the large lymphocytes—cells nearly as large as polynuclear leucocytes—predominate. Eosinophiles and nucleated red corpuscles are rare. Myelocytes are not present.

The pure myelogenous cases without associated enlargement of the spleen are rare. The most extreme hyperplasia of the bone marrow may exist without any tenderness. Occasionally the sternum, ribs, and flat bones show great irregularity and deformity, owing to definite tumor-like expansions.

Combined forms of leukæmia may occur, though they are not common. One such instance occurred at the Johns Hopkins Hospital. Here the spleen, marrow, and lymphatic glands all showed marked changes. The blood in this instance showed, besides a large proportion of lymphocytes and myelocytes, a considerable number of large mononuclear leucocytes.

Acute Leukæmia.—This is usually of the lymphatic type, and occurs in young persons. Fussell and Taylor have collected 56 cases from the literature. The fatal event occurs in two or three months.

Diagnosis.—The recognition of leukæmia can be determined only by microscopical examination of the blood. The clinical features may be identical with those of ordinary splenic anæmia, or of Hodgkin's disease. An interesting question arises whether real increase in the leucocytes is the only criterion of the existence of the disease. Thus, for instance, in the case whose chart is given on page 807, the patient came under observation in September, 1890, with 2,000,000 red blood-corpuscles per cubic millimetre, 30 per cent of hæmoglobin, and 500,000 white blood-corpuscles per cubic millimetre—a proportion of 1 to 4. As shown by the chart, throughout September, October, November, and December, this ratio was maintained. Early in January, under treatment with arsenic, the white corpuscles began to decrease, and gradually, as shown in the chart, the normal ratio was reached. At this time could it be said that the case was one of leukæmia without increase in the number of leucocytes? The blood examination by Ehrlich's method, as made by Thayer, showed that nucleated red corpuscles in large numbers as well as the characteristic myelocytes, elements which are but rarely found in normal blood, were

still present in numbers sufficient, at any rate, to suggest, if the patient had come under observation for the first time, that leukæmia might occur. By Ehrlich's method of blood examination a condition of leucocytosis can readily be distinguished from that of leukæmia, for in all ordinary leucocytoses the increase takes place solely in the polynuclear neutrophilic cells.

The remarkable "green cancer" or chloroma is, according to Dock, "a lymphomatous process similar in its classical features to leukæmia and pseudo-leukæmia."

Prognosis.—Recovery occasionally occurs. A great majority of the cases prove fatal within two or three years. Unfavorable signs are a tendency to hæmorrhage, persistent diarrhœa, early dropsy, and high fever. Remarkable variations are displayed in the course, and a transient improvement may take place for weeks or even months. The pure lymphatic form seems to be of particular malignancy, some cases proving fatal in from six to eight weeks; but there are exceptions, and I have recently seen a case in which the diagnosis was made ten years ago by W. H. Draper. The patient has had enlarged glands ever since, and, though not anæmic, the leucocytes were 242,000 per cubic millimetre, above 90 per cent of them being lymphocytes.

Treatment.—Fresh air, good diet, and abstention from mental worry and care, are the important general indications. The *indicatio morbi* cannot be met. There are certain remedies which have an influence upon the disease. Of these, arsenic, given in large doses, is the best. I have repeatedly seen improvement under its use. On the other hand, there are curious remissions in the disease which render therapeutical deductions very fallacious. I have seen such marked improvement without special treatment that the patient, from a bed-ridden, wretched condition, recovered strength enough to enable him to attend to light duties.

Quinine may be given in cases with a malarial history. Iron may be of value in some cases, as may also inhalations of oxygen.

Excision of the leukæmic spleen has been performed 24 times, with 1 recovery—the case of Franzolini. Fussell gives the statistics of 105 cases of splenectomy with 48 deaths. Of the cases of simple hypertrophy, 28 in number, 9 recovered. Of 16 cases of floating spleen, 15 recovered.

III. HODGKIN'S DISEASE.

Definition.—An affection characterized by progressive hyperplasia of the lymph-glands, with anæmia, and occasionally the development of secondary lymphoid growths in the liver, spleen, and other organs. The disease has also the names *pseudo-leukæmia*, *general lymphadenoma*, and *adénie*.

Hodgkin, the well-known morbid anatomist of Guy's Hospital, first described cases in detail, and by the labors of Wilks, Virchow, Billroth, and Cohnheim the disease attained definite recognition.

Etiology.—A majority of the cases are in young persons. In Gowers' table of 100 cases, 30 were under twenty years, 34 between twenty and forty, and 36 above forty. Three fourths of the cases are in males. In a

few instances heredity has been adduced as a possible cause, and antecedent disease, such as syphilis; but these are doubtful factors. More important is local irritation, upon which Trousseau lays special stress, and gives instances in which chronic irritation of the skin, chronic nasal catarrh, or the irritation of a decayed tooth gave rise to local gland swellings, which preceded a general development of the disease. In a large majority of the cases the disease comes on insidiously, without any recognizable cause.

Morbid Anatomy.—*The Lymph-glands.*—In a few cases the enlarged glands are hard and firm, but in a majority the growth is soft and elastic. In the early stage the individual glands are isolated, not larger than almonds or walnuts, and readily separated and movable. In more advanced stages the glands fuse together, and a group, as in the neck, may form a large tumor, the size of an orange or even of a cocoa-nut. About such masses the capsular tissues are hard and dense, forming a firm investment. A growth may perforate the capsule and invade contiguous parts, such as the muscles, skin, or the solid organs. On section, the tumor has a grayish-white appearance; it is smooth, and of variable consistence, either firm and dry or soft and juicy. Suppuration is most frequently seen when the growth reaches the skin. In the deep glands the formation of pus is rare. Caseation is not common; occasionally there are areas of necrosis very like it. The superficial glands are most often attacked, particularly the cervical groups, and they may be traced as continuous chains along the trachea and the carotids, uniting the axillary and mediastinal glands.

The axillary group is involved next in order of frequency, and the masses may pass beneath the pectorals and beneath the scapulæ. The inguinal glands occasionally form very large masses. Of the internal groups, those of the thorax are most often affected, either the chain in the posterior mediastinum or the bronchial group, or those of the anterior mediastinum. The trachea and the aorta with its branches may be completely surrounded by the growths, and be but little compressed. From the anterior mediastinum the masses may perforate the sternum and appear as an external tumor.

Of the abdominal groups, the retroperitoneal is most frequently involved and may form a continuous chain from the diaphragm to the inguinal canals, and extend into the pelvis. The glands may compress the ureters, involve the sacral or lumbar nerves, or impinge upon the iliac veins. Occasionally they adhere to the uterus and broad ligament so as to simulate fibroids. I saw, some years ago, one of the most distinguished gynæcologists of Germany perform laparotomy in a case of this kind, in which the diagnosis of myomatous tumors of the uterus had been made. Occasionally the mesenteric or hepatic lymph-glands may form large abdominal tumors.

Histologically the chief change is an increase in the cells, with or without thickening of the reticulum. In the early stage there is simple hyperplasia and the relations of the lymph paths are maintained, but when the glands are greatly enlarged the normal arrangement is disturbed. The reticulum varies extremely; in the softer growths it is expanded and can scarcely be found; in the harder structures the network of fibres is very

distinct, and there is probably an increased development of the adenoid tissue.

Spleen.—In 75 per cent of the cases collected by Gowers this organ was hypertrophied, and in 56 of these it presented lymphoid growths. The enlargement is rarely great, and does not approximate to that of the large leukæmic spleen. The lymphoid tumors form grayish-white bodies ranging in size from a pea to a walnut, and may resemble lymph-glands in appearance and consistence. Histologically, they consist of lymph corpuscles in a fibrous reticulum.

The marrow of the long bones may be converted into a rich lymphoid tissue; in a few instances the pyoid form, such as is more common in leukæmia, has been found. The tonsils may be involved and the follicles at the root of the tongue. Occasionally secondary growths are seen in the intestines.

The liver is often enlarged and may present scattered lymphoid tumors. The kidneys are occasionally involved and are the seat of growths similar to those in the spleen and liver. The lungs are occasionally directly attacked from the bronchial glands at the root, and secondary nodules may be found throughout their substance. Pleural effusions are not uncommon. Involvement of the nervous system is rare, but paraplegia may be induced by invasion of the spinal canal. The skin may be the seat of adenoid growths, as in a case reported by Greenfield.

Symptoms.—Enlargement of the glands of the neck, axilla, or groins is usually the first symptom noticed. In a few cases the anæmia and constitutional symptoms attract attention before the glandular involvement is evident. When the trouble begins in the deeper groups, pressure effects may be first noticed; thus, paroxysmal dyspnœa with pain in the chest may result from enlargement of the bronchial glands before any physical signs can be detected. Œdema of the feet and shooting pains in the nerves were the first symptoms in one case which I dissected for Ross, and in another case at the Montreal General Hospital there was paraplegia from pressure on the cord. Such instances, however, are exceptional, and in the majority of cases the swelling of the superficial glands is the earliest symptom. Epistaxis has occasionally been noted, but not so frequently as in leukæmia. With progressive enlargement of the glands the patient becomes anæmic.

Usually, the cervical group is first affected, and it may be impossible to decide whether the enlargement is syphilitic, tuberculous, or lymphadenomatous. One side is first affected as a rule, and it may be months or even, as in one of my cases, three years before the affection extends to other groups. Ultimately huge tumors may develop, which obliterate the neck and extend upon the shoulders and over the clavicles and sternum. The trachea is surrounded, great dyspnœa is produced, and not infrequently tracheotomy is necessary. In the later stages, the skin becomes involved and ulcerates. The axillary group may form large tumors, which compress the brachial or axillary veins and cause swelling of the arms. The inguinal glands may form large or even pendulous tumors.

In the thoracic glands, as mentioned, the various groups may be in-

volved and produce pressure upon the veins or upon the trachea. In a case recently under observation the superior cava was completely obliterated and a very extensive collateral circulation was established by means of the mammary and epigastric veins. The skin over the sternum was a mass of fluctuating veins, some of which contained phleboliths. In the abdomen the mesenteric glands may be enlarged, or more commonly the retroperitoneal group. When the patient is thin there may be no difficulty in detecting these, but in stout persons the diagnosis may be impossible. In connection with the affections of the abdominal glands there may be bronzing of the skin, which was well marked in Case IV of my series. A remarkable feature is the variations in the rate of growth and in the size of the glands. They may reduce rapidly and almost disappear from a region, and before death the number of those visible may diminish very much. The spleen may be enlarged and readily palpable. The thyroid also may be involved, and in a few instances the thymus has been affected. Though present in a majority of the cases, there may be enormous enlargement of the lymph-glands without marked anæmia. In one of my cases the blood-corpuscles did not sink below 4,000,000 per cubic millimetre, and in only one instance have I counted the blood below 2,000,000. The red blood-corpuscles rarely show extreme poikilocytosis. The white corpuscles may be moderately increased and the lymphocytes abundant, though usually there is little that is characteristic in the blood. Occasionally the leucocytes are greatly increased and the characters of the blood become those of a lymphatic leukæmia. Nucleated red blood-corpuscles may be present, but not in such numbers as in leukæmia.

Of cardiac symptoms, palpitation is common. Hæmic murmurs are often heard over the heart. Shortness of breath may be due to the anæmia, to pressure upon the trachea, or, in some instances, to pleuritic effusion associated with mediastinal growths. Fever is observed in nearly all cases; even in the early stages there is slight elevation. It may be of an irregular hectic type, or continuous, with evening exacerbation. Very remarkable are the cases with ague-like paroxysms, which may persist for weeks or months. They were present in Case I of my series. Pel, of Amsterdam, has given a thorough description of these attacks, and Ebstein has described a case under the remarkable title of Chronic Recurrent Fever, a New Infectious Disease. In his case during nine months the attacks were present for periods of from twelve to fourteen days and alternated with an apyrexia of ten or eleven days.

The digestive symptoms are usually not marked. It is not uncommon to find albumin in the urine. Headache, giddiness, and noises in the ear may be associated with the anæmia. Delirium and coma may ensue. Deafness may be produced by growth of the adenoid tissue in the pharynx close to the Eustachian tubes. Inequality of the pupils may be present, owing to pressure of the glands on the cervical sympathetic. The skin may show definite secondary lymphatic tumors, bronzing may occur, and occasionally a most intense and troublesome prurigo.

Diagnosis.—A tuberculous adenitis may at first be very difficult to differentiate. The chief points of distinction are as follows: Tuberculous

adenitis is more common in the young and involves the submaxillary group of glands more frequently than those of the anterior and posterior cervical triangles, which are usually affected first in Hodgkin's disease. The enlargement may last for years in a group without extending. The bunches are often, when small, welded together and, most important of all, tend to suppurate—a feature rarely seen in true lymphadenoma, except when it has attained very large size. Strict limitation to one side of the neck or to the axilla is suggestive of tuberculous disease rather than lymphadenoma.

There is an acute tuberculous adenitis, which may involve the lymph-glands of the neck, producing enormous enlargement. A man, aged twenty-four, was admitted to the General Hospital, Montreal, with great swelling of the cervical glands on both sides, tonsillitis, and sloughing pharyngitis, with irregular fever and diarrhœa. The case was at first regarded as one of Hodgkin's disease. The occurrence of rigors and intermittent pyrexia is in favor of lymphadenoma. There are cases in which it may for a time be impossible to make a diagnosis. When the glands are only moderately enlarged on one side of the neck or axilla, they should be removed, and the diagnosis can then be thoroughly established.

Prognosis.—Recovery is very rare. The course of the disease is extremely variable. Early and rapid growth in the mediastinal groups may produce pressure effects and cause death before the development is extreme. In some cases the enlargements spread rapidly and group after group becomes involved in a few months. These acute case may run a course in three or four months. Chronic cases may last for three or four years. Periods of quiescence are not uncommon. The tumors may not only cease to grow, but gradually diminish and even disappear, without special treatment. Usually a cachexia develops, the anæmia progresses, and there are dropsical symptoms. The mode of death is usually by asthenia; less commonly by pressure from a tumor; and occasionally in coma.

Treatment.—When small and localized the glands should be removed. Local applications are of doubtful benefit. I have never seen special improvement follow the persistent use of iodine or the various ointments.

Arsenic has a positive value in the disease. It should be given in increasing doses, and stopped when unpleasant effects are manifested. The results have in many instances been striking. Due allowance must be made for the fluctuations in the size of the growths which occur spontaneously. Ill effects from the administration of Fowler's solution, even for months at a time, are rare, but I have had a case in which neuritis followed the use of ℥ iv ʒ j ♏xviij within a period of less than three months. Recoveries have been reported under this treatment. Personally, no instance of recovery has come under my notice in the cases of which I have notes. Phosphorus is recommended by Gowers and Broadbent, and should be used if the arsenic is not well borne. Quinine, iron, and cod-liver oil are useful as tonics. Every possible means must be taken to support the patient's strength.

IV. PURPURA.

Strictly speaking, purpura is a symptom, not a disease; but under this term are conveniently arranged a number of affections characterized by extravasations of the blood into the skin. In the present state of our knowledge a satisfactory classification cannot be made. Excluding symptomatic purpura, W. Koch groups all forms, including hæmophilia, under the designation *hæmorrhagic diathesis*, believing that intermediate forms link the mild purpura simplex and the most intense purpura hæmorrhagica; while F. A. Hoffmann considers them all (except hæmophilia) under the heading *morbus maculosus*. The purpuric spots vary from 1 to 3 or 4 mm. in diameter. When small and pin-point-like they are called petechiæ; when large, they are known as ecchymoses. At first bright red in color, they become darker, and gradually fade to brownish stains. They do not disappear on pressure.

In all cases of purpura the coagulation time of the blood should be estimated (Wright); the coagulometer is a useful clinical instrument for the purpose. Normal blood clots in the tubes in from three to five minutes. In some forms of purpura the coagulation time is retarded to ten or fifteen minutes, and in hæmophilia it has been delayed to fifty minutes.

The following is a provisional grouping of the cases:

Symptomatic Purpura.—(*a*) **Infectious.**—In pyæmia, septicæmia, and malignant endocarditis (particularly in the last affection), ecchymoses may be very abundant. In typhus fever the rash is always purpuric. Measles, scarlet fever, and more particularly small-pox, have each a variety characterized by an extensive purpuric rash.

(*b*) **Toxic.**—The virus of snakes produces with great rapidity extravasation of blood—a condition which has been very carefully studied by Weir Mitchell. Certain medicines, particularly copaiba, quinine, belladonna, mercury, ergot, and the iodides occasionally, are followed by a petechial rash. Purpura may follow the use of comparatively small doses of iodide of potassium. It is not a very common occurrence, considering the great frequency with which the drug is employed. A fatal event may be caused by a small amount, as in a case reported by Stephen Mackenzie of a child which died after a dose of 2½ grains. An erythema may precede the hæmorrhage. It is not always a simple purpura, but may be an acute febrile eruption of great intensity. In September, 1894, a man aged forty-eight was admitted under my care with arterio-sclerosis and dropsy. The latter yielded rapidly to digitalis and diuretin. When convalescent he was ordered iodide of potassium in 10-grain doses three times a day, and took in fourteen days 420 grains. He had high fever, coryza, swelling of the throat, and the most extensive purpura over the whole body. Under this division, too, comes the purpura so often associated with jaundice.

(*c*) **Cachectic.**—Under this heading are best described the instances of purpura which develop in the constitutional disturbance of cancer, tuberculosis, Hodgkin's disease, Bright's disease, scurvy, and in the debility of old age. In these cases the spots are usually confined to the extremities.

They may be very abundant on the lower limbs and about the wrists and hands. This constitutes, probably, the commonest variety of the disease, and many examples of it can be seen in the wards of any large hospital.

(*d*) **Neurotic.**—One variety is met with in cases of organic disease. It is the so-called myelopathic purpura, which is seen occasionally in locomotor ataxia, particularly following attacks of the lightning pains and, as a rule, involving the area of the skin in which the pains have been most intense. Cases have been met with also in acute myelitis and in transverse myelitis, and occasionally in severe neuralgia. Another form is the remarkable hysterical condition in which stigmata, or bleeding points, appear upon the skin.

(*e*) **Mechanical.**—This variety is most frequently seen in venous stasis of any form, as in the paroxysms of whooping-cough and in epilepsy.

Arthritic.—This form is characterized by involvement of the joints. It is usually known, therefore, as rheumatic, though in reality the evidence upon which this view is based is not conclusive. Of 200 cases of purpura analyzed by Stephen Mackenzie, 61 had a history of rheumatism. For the present it seems more satisfactory to use the designation arthritic. Three groups of cases may be recognized:

(*a*) A mild form, often known as **Purpura simplex,** seen most commonly in children, in whom, with or without articular pain, a crop of purpuric spots appears upon the legs, less commonly upon the trunk and arms. As pointed out by Graves, this form is not infrequently associated with diarrhœa. The disease is seldom severe. There may be loss of appetite, and slight anæmia. Fever is not, as a rule, present, and the patients get well in a week or ten days. These cases are usually regarded as rheumatic, and are certainly associated, in some instances, with undoubted rheumatic manifestations; yet in a majority of the patients which I have seen the arthritis was slighter than in the ordinary rheumatism of children, and no other manifestations were present.

(*b*) **Purpura (Peliosis) rheumatica** (*Schönlein's Disease*).—This remarkable affection is characterized by multiple arthritis, and an eruption which varies greatly in character, sometimes *purpuric*, more commonly associated with *urticaria* or with *erythema exudativum*. The disease is most common in males between the ages of twenty and thirty. It not infrequently sets in with sore throat, a fever from 101° to 103°, and articular pains. The rash, which makes its appearance first on the legs or about the affected joints, may be a simple purpura or may show ordinary urticarial wheals. In other instances there are nodular infiltrations, not to be distinguished from erythema nodosum. The combination of wheals and purpura, the *purpura urticans*, is very distinctive. Much more rarely vesication is met with, the so-called *pemphigoid purpura*. The amount of œdema is variable; occasionally it is excessive. In one case, which I saw in Montreal with Molson, the chin and lower lip were enormously swollen, tense, glazed, and deeply ecchymotic. The eyelids were swollen and purpuric, while scattered over the cheeks and about the joints were numerous spots of purpura urticans. These are the cases which have been described as *febrile*

purpuric œdema. The temperature range, in mild cases, is not high, but may reach 102° or 103°.

The urine is sometimes reduced in amount and may be albuminous. The joint affections are usually slight, though associated with much pain, particularly as the rash comes out. Relapses may occur and the disease may return at the same time for several years in succession.

The diagnosis of Schönlein's disease offers no difficulty. The association of multiple arthritis with purpura and urticaria is very characteristic. In a case which I saw with Musser there was endo-pericarditis, and the question at first arose whether the patient had malignant endocarditis with extensive cutaneous infarcts.

Schönlein's peliosis is thought by most writers to be of rheumatic origin, and certainly many of the cases have the characters of ordinary rheumatic fever, *plus* purpura. By many, however, it is regarded as a special affection, of which the arthritis is a manifestation analogous to that which occurs in hæmophilia and in scurvy. The frequency with which sore throat precedes the attack, and the occasional occurrence of endocarditis or pericarditis, are certainly very suggestive of true rheumatism.

The cases usually do well, and a fatal event is extremely rare. The throat symptoms may persist and give trouble. In two instances I have seen necrosis and sloughing of a portion of the uvula.

(*c*) **Henoch's Purpura.**—This variety, seen chiefly in children, is characterized by (1) relapses or recurrences, often extending over several years; (2) cutaneous lesions, which are those of erythema multiforme rather than of simple purpura; (3) gastro-intestinal crises—pain, vomiting, and diarrhœa; (4) joint pains or swelling, often trifling; (5) hæmorrhages from the mucous membranes. When from the kidney, an intense hæmorrhagic nephritis may supervene, which proved fatal, with the symptoms of acute Bright's disease, in one of my cases, and became chronic in a case under D. W. Prentiss. Any one or two of the above symptoms may be absent; the intestinal crises with enlargement of the spleen may be present and recur for months before the true nature of the trouble becomes manifest. This form has an interesting connection with the angio-neurotic œdema, which is also characterized by severe gastro-intestinal crises. The prognosis is, as a rule, good; 3 of the 11 cases which I have reported died.*

Purpura Hæmorrhagica.—Under this heading may be considered the cases of very severe purpura with hæmorrhages from the mucous membranes. The affection, known as the *morbus maculosus* of Werlhof, is most commonly met with in young and delicate individuals, particularly in girls; but cases are described in which the disease has attacked adults in full vigor. After a few days of weakness and debility, purpuric spots appear on the skin and rapidly increase in numbers and size. Bleeding from the mucous surfaces sets in, and the epistaxis, hæmaturia, and hæmoptysis may cause profound anæmia. Chart XXI illustrates the rapidity with which anæmia is produced and the gradual recovery. Death may take place from loss of blood, or from hæmorrhage into the brain. Slight

* Am. Jour. of the Med. Sciences, December, 1895.

fever usually accompanies the disease. In favorable cases the affection terminates in from ten days to two weeks. There are instances of purpura hæmorrhagica of great malignancy, which may prove fatal within twenty-four hours—*purpura fulminans*. This form is most commonly met with in children, and is characterized by cutaneous hæmorrhages, which develop with great rapidity. Death may occur before any bleeding takes place from the mucous membranes.

In the *diagnosis* of purpura hæmorrhagica it is important to exclude scurvy, which may be done by the consideration of the previous health,

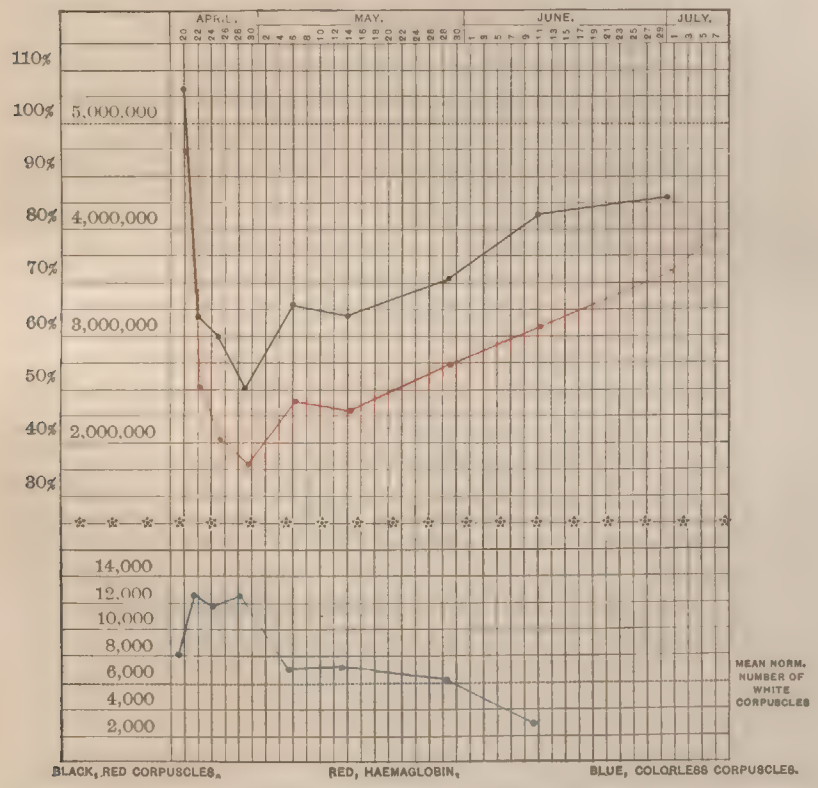

CHART XXI.—Illustrates the rapidity with which anæmia is produced in purpura hæmorrhagica and the gradual recovery.

the circumstances under which the disease develops, and by the absence of swelling of the gums. The malignant forms of the fevers, particularly small-pox and measles, are distinguished by the prodromes and the higher temperature.

Treatment.—In symptomatic purpura attention should be paid to the conditions under which it develops, and measures should be employed to increase the strength and to restore a normal blood condition. Tonics, good food, and fresh air meet these indications. In the simple purpura of

children, or that associated with slight articular trouble, arsenic in full doses should be given. No good is obtained from the small doses, but the Fowler's solution should be pushed freely until physiological effects are obtained. In peliosis rheumatica the sodium salicylates may be given, but with discretion. I confess not to have seen any special control of the hæmorrhages by this remedy.

Aromatic sulphuric acid, ergot, turpentine, acetate of lead, or tannic and gallic acids, may be used, and in some instances they seem to check the bleeding. Oil of turpentine is perhaps the best remedy, in 10 or 15 minims doses three or four times a day. Wright, of Netley, advises the use of calcium chloride in 20-grain doses four times a day (for three or four days) to increase the coagulability of the blood. In bleeding from the mouth, gums, and nose, the inhalation of the carbon dioxide is sometimes useful. The rinsing of the mouth with gelatin has been recommended.

HÆMORRHAGIC DISEASES OF THE NEW-BORN.

1. **Syphilis Hæmorrhagica Neonatorum.**—The child may be born healthy, or there may be signs of hæmorrhage at birth. Then in a few days there are extensive cutaneous extravasations and bleeding from the mucous surfaces and from the navel. The child may become deeply jaundiced. The post mortem shows numerous extravasations in the internal organs and extensive syphilitic changes in the liver and other organs.

2. **Epidemic Hæmoglobinuria** (*Winckel's Disease*).—Hæmoglobinuria in the new-born, which occasionally develops in epidemic form in lying-in institutions, is a very fatal affection, which sets in usually about the fourth day of life. The child becomes jaundiced, and there are marked gastro-intestinal symptoms, with fever, jaundice, rapid respiration, and sometimes cyanosis. The urine contains albumin and blood-coloring matter—methæmoglobin. The disease has to be distinguished from the simple icterus neonatorum, with which there may sometimes be blood or blood-coloring matter in the urine. The post mortem shows an absence of any septic condition of the umbilical vessels, but the spleen is swollen, and there are punctiform hæmorrhages in different parts. Some cases have shown in a marked degree acute fatty degeneration of the internal organs—the so-called Buhl's disease.

3. **Morbus Maculosus Neonatorum.**—Apart from the common visceral hæmorrhages, the result of injuries at birth, bleeding from one or more of the surfaces is a not uncommon event in the new-born, particularly in hospital practice. Forty-five cases occurred in 6,700 deliveries (C. W. Townsend). The bleeding may be from the navel alone, but more commonly it is general. Of Townsend's 50 cases, in 20 the blood came from the bowels (*melæna neonatorum*), in 14 from the stomach, in 14 from the mouth, in 12 from the nose, in 18 from the navel, in 3 from the navel alone. The bleeding begins within the first week, but in rare instances is delayed to the second or third. Thirty-one of the cases died and 19 recovered. The disease is usually of brief duration, death occurring in from one to seven days. The temperature is often elevated. The nature

The nature of the disease is unknown. An increase in the number of the red blood corpuscles - erythrocythaemia - ~~has been~~ with a peculiar frailty of the blood vessels has been supposed. A deficiency of the leucocytes and a deficiency of the blood plates have been noted, though in a case from my clinic studied by Steiner these structures were normal. Wright has found the coagulation time much retarded, as long as 23 and 46 minutes

is considered at length

described

Hegg; 4: 1 Stempel

The German cases are chiefly collected: of 209 cases collected within the ten years 1890-1900 by Stempel 96 were German, 93 English or American and only 3 or 6 French. Hungarian & Russian cases are reported - rarely - ~~France~~ in the negro often my clinic

of the disease is unknown. As a rule, nothing abnormal is found post mortem. The general and not local nature of the affection, its self-limited character, the presence of fever, and the greater prevalence of the disease in hospitals, suggest an infectious origin (Townsend). The bleeding may be associated with intense hæmatogenous jaundice. Not every case of bleeding from the stomach or bowels belongs in this category. Ulcers of the œsophagus, stomach, and duodenum have been found in the new-born dead of *melæna neonatorum*. The child may draw the blood from the breast and subsequently vomit it. In the treatment the external warmth must be maintained, and in feeble infants the *couveuse* may be used. Camphor is recommended and ergotin hypodermically.

V. HÆMOPHILIA.

Definition.—An hereditary, constitutional fault, characterized by a tendency to uncontrollable bleeding, either spontaneous or from slight wounds, sometimes associated with a form of arthritis. The coagulation time of the blood is usually much retarded.

Early in the century several physicians of this country called attention to the occurrence of profuse hæmorrhage from slight causes. The fact that fatal hæmorrhage might occur from slight, trifling wounds had been known for centuries. The recognition of the family nature of the disease is due to the writings of Buel, Otto, Hay, Coates, and others in this country. The disease has been elaborately treated in the monographs of Legg and Grandidier.

Etiology.—In a majority of cases the disposition is hereditary. The fault may be acquired, however, but nothing is known of the conditions under which the disease may thus arise in healthy stock.

The hereditary transmission in this disease is remarkable. In the Appleton-Swain family, of Reading, Mass., there have been cases for nearly two centuries; and F. F. Brown, of that town, tells me that instances have already occurred in the seventh generation. The usual mode of transmission is through the mother, who is not herself a bleeder, but the daughter of one. Atavism through the female alone is almost the rule, and the daughters of a bleeder, though healthy and free from any tendency, are almost certain to transmit the disposition to the male offspring. The affection is much more common in males than in females, the proportion being estimated at 11 to 1, or even 13 to 1. The tendency usually appears within the first two years of life. It is rare for manifestations to be delayed until the tenth or twelfth year. Families in all conditions of life are affected. The bleeder families are usually large. The members are healthy-looking, and have fine, soft skins.

Morbid Anatomy.—No special peculiarities have been described. In some instances changes have been found in the smaller vessels; but in others careful studies have been negative. An unusual thinness of the vessels has been noted. Hæmorrhages have been found in and about the capsules of the joints, and in a few instances inflammation of the synovial

surfaces. The nature of the disease is undetermined, and we do not yet know whether it depends upon a peculiar frailty of the blood-vessels or some peculiarity in the constitution of the blood, which prevents the normal thrombus formation in a wound.

Symptoms.—Usually hæmophilia is not noted in the child until a trifling cut is followed by serious or uncontrollable hæmorrhage, or spontaneous bleeding occurs and presents insuperable difficulties in its arrest. The symptoms may be grouped under three divisions: external bleedings, spontaneous and traumatic; interstitial bleedings, petechiæ and ecchymoses; and the joint affections. The external bleedings may be spontaneous, but more commonly they follow cuts and wounds. In 334 cases (Grandidier) the chief bleedings were epistaxis, 169; from the mouth, 43; stomach, 15; bowels, 36; urethra, 16; lungs, 17; and in a few instances bleeding from the skin of the head, the tongue, finger-tips, tear-papilla, eyelids, external ear, vulva, navel, and scrotum.

Traumatic bleeding may result from blows, cuts, scratches, etc., and the blood may be diffused into the tissues or discharged externally. Trivial operations have proved fatal, such as the extraction of teeth, circumcision, or venesection. It is possible that there may be local defects which make bleeding from certain parts of the body more dangerous. D. Hayes Agnew mentioned to me the case of a bleeder who had always bled from cuts and bruises above the neck, never from those below. The bleeding is a capillary oozing. It may last for hours, or even many days. Epistaxis may prove fatal in twenty-four hours. In the slow bleeding from the mucous surfaces large blood tumors may form and project from the nose or mouth, forming remarkable-looking structures, and showing that the blood has the power of coagulation. The interstitial hæmorrhages may be spontaneous, or may result from injury. Petechiæ or large extravasations—hæmatomata—may occur, the latter usually following blows.

The joint affections of hæmophilia are remarkable. There may simply be pain, or attacks which come on suddenly with fever, and closely resemble acute rheumatism. The larger joints are usually affected. Arthritis may usher in an attack of hæmorrhage.

So far as the examination of the blood goes, no changes of special moment have been noted. When the bleeding has been severe, it is thin and watery, but at the beginning of the bleeding the blood is rich in corpuscles and coagulates firmly.

Diagnosis.—In the diagnosis of the condition the family tendency is important. A single uncontrollable hæmorrhage in child or adult is not to be ranked as hæmophilia; but it is only when a person shows a marked tendency to multiply hæmorrhages, spontaneous or traumatic, which tendency is not transitory but persists, and particularly if there have been joint affections, that we may consider the condition hæmophilia. Such conditions as epistaxis, recurring for years—if no other hæmorrhage occurs—or recurring hæmaturia from one kidney, which has been spoken of as unilateral renal hæmophilia, have no association with the true disease. Peliosis rheumatica is an affection which touches hæmophilia very closely, particularly in the relation of the joint swellings. It may also show itself

The blood platelets are absent or scanty
The coagulation time may be retarded

Joint affections. - The knees and elbows are chiefly involved, but the small joints may be attacked. The onset is usually acute, with slight fever and swelling and pain and sometimes redness in the joints. In other instances there is haemorrhagic effusion without fever. Young recognizes three stages; first, haemarthrosis; secondly, an inflammatory process with fever and spindle-formed swelling, which is apt to be mistaken for tuberculosis; and lastly there may be extensive organic changes in the joints, which may even resemble those of arthritis deformans.

Historical.
Distribution
Ch. 1897.

Ice Harbor, Upper work.

Seems "the calamity factor" as I has
been well named.

in several members of a family. The diagnosis from the various forms of purpura is usually easy.

Prognosis.—The patients rarely die in the first bleeding. The younger the individual the worse is the outlook, though it is rarely fatal in the first year. Grandidier states that of 152 boy subjects, 81 died before the termination of the seventh year. The longer the bleeder survives the greater the chance of his outliving the tendency; but it may persist to old age, as shown in the case of Oliver Appleton, the first reported American bleeder, who died at an advanced age of hæmorrhage from a bed-sore and from the urethra. The prognosis is graver in a boy than in a girl. In the latter menstruation is sometimes early and excessive, but fortunately, in the female members of hæmophilic families, neither this function nor the act of parturition brings with it special dangers.

Treatment.—Members of a bleeder's family, particularly the boys, should be guarded from injury, and operations of all sorts should be avoided. The daughters should not marry, as it is through them that the tendency is propagated.

When an injury or wound has occurred, absolute rest and compression should first be tried, and if these fail the styptics may be used. In epistaxis ice, tannic and gallic acid may be tried before resorting to plugging. Internally ergot seems to have done good in several cases. Legg advises the perchloride of iron in half-drachm doses every two hours with a purge of sulphate of soda. For the epistaxis ~~of the disease~~ the inhalation of carbon dioxide through the nostrils, as recommended by A. E. Wright, ~~may be tried~~. He also recommends a solution of fibrin ferment and chloride of calcium as a styptic. ~~Biendwald has reported a case of~~ a ~~child in which~~ the application of fresh blood to the wound checked the bleeding after all other means had failed. Gelatin in 5-per-cent solution is warmly recommended. Venesection has been tried in several cases. Transfusion has been employed, but without success. During convalescence, iron and arsenic should be freely used.

VI. SCURVY (*Scorbutus*).

Definition.—A constitutional disease characterized by great debility, with anæmia, a spongy condition of the gums, and a tendency to hæmorrhages.

Etiology.—The disease has been known from the earliest times, and has prevailed particularly in armies in the field and among sailors on long voyages.

From the early part of this century, owing largely to the efforts of Lind and to a knowledge of the conditions upon which the disease depends, scurvy has gradually disappeared from the naval service. In the mercantile marine, cases still occasionally occur, owing to the lack of proper and suitable food.

The disease develops whenever individuals have subsisted for prolonged periods upon a diet in which fresh vegetables or their substitutes

are lacking. An insufficient diet appears to be an essential element in the disease, and all observers are now unanimous that it is the absence of those ingredients in the food which are supplied by fresh vegetables. What these constituents are has not yet been definitely determined. Garrod holds that the defect is in the absence of the potassic salts. Others believe that the essential factor is the absence of the organic salts present in fruits and vegetables. Ralfe, who has made a very careful study of the subject, believes that the absence from the food of the malates, citrates, and lactates reduces the alkalinity of the blood, which depends upon the carbonate directly derived from these salts. This diminished alkalinity, gradually produced in the scurvy patients, is, he believes, identical with the effect which can be artificially produced in animals by feeding them with an excess of acid salts; the nutrition is impaired, there are ecchymoses, and profound alterations in the characters of the blood. The acidity of the urine is greatly reduced and the alkaline phosphates are diminished in amount. One of the most interesting of recent facts relating to scurvy has been the great frequency of it in children, in connection, as a rule, with improper diet. It will be referred to more fully in a subsection.

In opposition to this chemical view it has been urged that the disease really depends upon a specific (as yet unknown) micro-organism.

In the United States scurvy has become a very rare disease. To the hospitals in the seaport towns sailors are now and then admitted with it. In large almshouses outbreaks occasionally occur. A very great increase of foreign population of a low grade has in certain districts made the disease not at all uncommon. In the mining districts of Pennsylvania the Hungarian, Bohemian, and Italian settlers are not infrequently attacked. McGrew has recently reported 42 cases in Chicago, limited entirely to Poles. He ascertained that in a large proportion of the cases the diet was composed of bread, strong coffee, and meat. Occasionally one meets with scurvy among quite well-to-do people. One of the most characteristic cases I have ever seen was in a woman with chronic dyspepsia, who had lived for many months chiefly on tea and bread. Some years ago scurvy was not infrequent in the large lumbering camps in the Ottawa Valley. Judging from the Report of the American Pædiatric Society, we must infer that infantile scurvy is on the increase in this country.

In parts of Russia scurvy is endemic, at certain seasons reaching epidemic proportions; and the leading authorities upon the disorder, now in that country, are almost unanimous, according to Hoffmann,* in regarding it as infectious.

Other factors play an important part in the disease, particularly physical and moral influences—overcrowding, dwelling in cold, damp quarters, and prolonged fatigue under depressing influences, as during the retreat of an army. Among prisoners, mental depression plays an important *rôle*. It is stated that epidemics of the disease have broken out in the French convict-ships *en route* to New Caledonia even when the diet was amply

* Lehrbuch der Constitutionskrankheiten, F. A. Hoffmann (1893), a work to which the student is referred for the best exposition of this group of disorders.

In Gt Britain & Ireland it has become very rare. Only
302 cases were admitted to the Lancashire hospital in the
22 years ending 1846 (Reports in Growth) In the Registrar
General's report for 1847 there were only 2 deaths from
scurvy.

sufficient. Nostalgia is sometimes an important element. It is an interesting fact that prolonged starvation in itself does not necessarily cause scurvy. Not one of the professional fasters of late years has displayed any scorbutic symptom. The disease attacks all ages, but the old are more susceptible to it. Sex has no special influence, but during the siege of Paris it was noted that the males attacked were greatly in excess of the females.

Morbid Anatomy.—The anatomical changes are marked, though by no means specific, and are chiefly those associated with hæmorrhage. The blood is dark and fluid. The microscopical alterations are those of a severe anæmia, without leucocytosis. The bacteriological examination has not yielded anything very positive. Practically there are no changes in the blood, either anatomical or chemical, which can be regarded as peculiar to the disease. The skin shows the ecchymoses evident during life. There are hæmorrhages into the muscles, and occasionally about or even into the joints. Hæmorrhages occur in the internal organs, particularly on the serous membranes and in the kidneys and bladder. The gums are swollen and sometimes ulcerated, so that in advanced cases the teeth are loose and have even fallen out. Ulcers are occasionally met with in the ileum and colon. Hæmorrhages into the mucous membranes are extremely common. The spleen is enlarged and soft. Parenchymatous changes are constant in the liver, kidneys, and heart.

Symptoms.—The disease is insidious in its onset. Early symptoms are loss in weight, progressively developing weakness, and pallor. Very soon the gums are noticed to be swollen and spongy, to bleed easily, and in extreme cases to present a fungous appearance. These changes, regarded as characteristic, are sometimes absent. The teeth may become loose and even fall out. Actual necrosis of the jaw is not common. The breath is excessively foul. The tongue is swollen, but may be red and not much furred. The salivary glands are occasionally enlarged. Hæmorrhages beneath the mucous membranes of the mouth are common. The skin becomes dry and rough, and ecchymoses soon appear, first on the legs and then on the arms and trunk, and particularly into and about the hair-follicles. They are petechial, but may become larger, and when subcutaneous may cause distinct swellings. In severe cases, particularly in the legs, there may be effusion between the periosteum and the bone, forming irregular nodes, which, in the case of a sailor from a whaling vessel who came under my observation, had broken down and formed foul-looking sores. The slightest bruise or injury causes hæmorrhages into the injured part. Œdema about the ankles is common. The "scurvy sclerosis," seen oftenest in the legs, is a remarkable infiltration of the subcutaneous tissues and muscles, forming a brawny induration, the skin over which may be blood-stained. Hæmorrhages from the mucous membranes are less constant symptoms; epistaxis is, however, frequent. Hæmoptysis and hæmatemesis are uncommon. Hæmaturia and bleeding from the bowels may be present in very severe cases.

Palpitation of the heart and feebleness and irregularity of the impulse are prominent symptoms. A hæmic murmur can usually be heard at the

base. Hæmorrhagic infarction of the lungs and spleen has been described. Respiratory symptoms are not common. The appetite is impaired, and owing to the soreness of the gums the patient is unable to chew the food. Constipation is more frequent than diarrhœa. Pain, tenderness, or swelling in the joints were present in 13 of McGrew's 42 cases. The urine is often albuminous. The changes in its composition are not constant; the specific gravity is high; the color is deeper; and the phosphates are increased. The statements with reference to the inorganic constituents are contradictory. Some say the phosphates and potash are deficient; others that they are increased.

There are mental depression, indifference, in some cases headache, and in the later stages delirium. Cases of convulsions, of hemiplegia, and of meningeal hæmorrhage have been described. Remarkable ocular symptoms are occasionally met with, such as night-blindness or day-blindness.

In advanced cases necrosis of the bones may occur, and in young persons even separation of the epiphyses. There are instances in which the cartilages have separated from the sternum. The callus of a recently repaired fracture has been known to undergo destruction. Fever is not present, except in the later stages, or when secondary inflammations in the internal organs appear. The temperature may, indeed, be sometimes below normal. Acute arthritis is an occasional complication.

Diagnosis.—No difficulty is met in the recognition of scurvy when a number of persons are affected together. In isolated cases, however, the disease is distinguished with difficulty from certain forms of purpura. The association with manifest insufficiency in diet, and the rapid amelioration with suitable food, are points by which the diagnosis can be readily settled.

Prognosis.—The outlook is good, unless the disease is far advanced and the conditions persist which lead to its development. The mortality now is rarely great. Death results from gradual heart-failure, occasionally from sudden syncope. Meningeal hæmorrhage, extravasation into the serous cavities, entero-colitis, and other intercurrent affections may prove fatal.

Prophylaxis.—The regulations of the Board of Trade require that a sufficient supply of antiscorbutic articles of diet be taken on each ship; so that now, except as the result of accident, the occurrence of scurvy is rare in sailors.

Treatment.—The juice of two or three lemons daily and a varied diet, with plenty of fresh vegetables, suffice to cure all cases of scurvy, unless far advanced. When the stomach is much disordered, small quantities of scraped meat and milk should be given at short intervals, and the lemon-juice in gradually increasing quantities. A bitter tonic, or a steel and bark mixture, may be given. As the patient gains in strength, the diet may be more liberal, and he may eat freely of potatoes, cabbage, water-cresses, and lettuce. The stomatitis is the symptom which causes the greatest distress. The permanganate of potash or dilute carbolic acid forms the best mouth-wash. Pencilling the swollen gums with a tolerably strong solution of nitrate of silver is very useful. The solution is better than the solid stick,

as it reaches to the crevices between the granulations. The constipation which is so common is best treated with large enemata. For other conditions, such as hæmorrhages and ulcerations, suitable measures must be employed.

INFANTILE SCURVY (*Barlow's Disease*).

As in adults, scurvy may occur in children in consequence of imperfect food supply.

W. B. Cheadle and Gee, in London, have described in very young children a cachexia associated with hæmorrhage. Cheadle regarded the cases as scurvy ingrafted on a rickety stock. Gee called his cases periosteal cachexia. Cases had previously been regarded as acute rickets.

A few years later Barlow made an exhaustive study of the condition with careful anatomical observations. The affection is now recognized as infantile scurvy, and in Germany is called Barlow's Disease. The American Pædiatric Society has collected (1898) in this country 379 cases. Of these, the hygienic surroundings were good in 303. A majority of the patients were under twelve months. The proprietary foods, particularly malted milk and condensed milk, seem to be the most important factors in producing the disease. There are instances in which it has developed in breast-fed infants, and in others fed on the carefully prepared milk of the Walker-Gordon laboratories.

The following is a general clinical summary, taken from Barlow's Bradshaw Lecture, 1894:

"So long as it is left alone the child is tolerably quiet; the lower limbs are kept drawn up and still; but when placed in its bath or otherwise moved there is continuous crying, and it soon becomes clear that the pain is connected with the lower limbs. At this period the upper limbs may be touched with impunity, but any attempt to move the legs or thighs gives rise to screams. Next, some obscure swelling may be detected, first on one lower limb, then on the other, though it is not absolutely symmetrical. . . . The swelling is ill-defined, but is suggestive of thickening round the shafts of the bones, beginning above the epiphyseal junctions. Gradually the bulk of the limbs affected becomes visibly increased. . . . The position of the limbs becomes somewhat different from what it was at the outset. Instead of being flexed they lie everted and immobile, in a state of pseudo-paralysis. . . . About this time, if not before, great weakness of the back becomes manifest. A little swelling of one or both scapulæ may appear, and the upper limbs may show changes. These are rarely so considerable as the alterations in the lower limbs. There may be swelling above the wrists, extending for a short distance up the forearm, and some swelling in the neighborhood of the epiphyses of the humerus. There is symmetry of lesions, but it is not absolute; and the limb affection is generally consecutive, though the involvement of one limb follows very close upon another. The joints are free. In severe cases another symptom may not be found—namely, crepitus in the regions adjacent to the junctions of the shafts with the epiphyses. The upper and lower extremities of the femur, and the upper extremity of the tibia, are the common

sites of such fractures; but the upper end of the humerus may also be so affected. . . . A very startling appearance may be observed at this period in the front of the chest. The sternum, with the adjacent costal cartilages and a small portion of the contiguous ribs, seems to have sunk bodily back, *en bloc*, as though it had been subjected to some violence which had fractured several ribs in the front and driven them back. Occasionally thickenings of varying extent may be found on the exterior of the vault of the skull, or even on some of the bones of the face. . . . Here also must be mentioned a remarkable eye phenomenon. There develops a rather sudden proptosis of one eyeball, with puffiness and very slight staining of the upper lid. Within a day or two the other eye presents similar appearances, though they may be of less severity. The ocular conjunctiva may show a little ecchymosis, or may be quite free. With respect to the constitutional symptoms accompanying the above series of events the most important feature is the profound anæmia which is developed. . . . The anæmia is proportional to the amount of limb involvement. As the case proceeds, there is a certain earthy-colored or sallow tint, which is noteworthy in severe cases, and when once this is established bruise-like ecchymoses may appear, and more rarely small purpuræ. Emaciation is not a marked feature, but asthenia is extreme and suggestive of muscular failure. The temperature is very erratic; it is often raised for a day or two, when successive limbs are involved, especially during the tense stage, but is rarely above 101° or 102°. At other times it may be normal or subnormal." If the teeth have appeared the gums may be spongy.

The condition must always be looked for in young children with difficulty in moving the lower limbs, or in whom paralysis is suspected. What is known sometimes as Parrot's disease, or syphilitic pseudo-paralysis, may be confounded with it. In it the loss of motion is more or less sudden in the upper or lower limbs, or in both, due to a solution of continuity and separation of the cartilage at the end of the diaphysis. There are usually crepitation and much pain on movement.

The essential lesion is a subperiosteal blood extravasation, which causes the thickening and tenderness in the shafts of the bones. In some instances there is hæmorrhage in the intramuscular tissue.

The prophylaxis is most important. The various proprietary forms of condensed milk and preserved foods for infants should not be used. The fresh cow's milk should be substituted, and a teaspoonful of meat-juice or gravy may be given with a little mashed potato. Orange-juice or lemon-juice should be given three or four times a day. Recovery is usually prompt and satisfactory.

VII. STATUS LYMPHATICUS. LYMPHATISM.

Much attention has been paid lately to a somewhat rare condition met with chiefly in children and young persons, in which the lymphatic glands and lymph tissues throughout the body, the spleen, the thymus, and the lymphoid bone marrow are in a state of hyperplasia. These features have

been found associated with rickets and with hypoplasia of the heart and aorta. The special interest lies in the fact that these pathological conditions have been met with frequently in cases of sudden death. Paltauf and others of the Vienna school, who have written extensively on the subject, believe that individuals with this hyperplasia have lowered powers of resistance, and are particularly liable to paralysis of the heart. The condition has not received much attention in England and in this country. An excellent account of it, by James Ewing, appeared in the New York Medical Journal of July 10, 1897.

Anatomical Condition.—(*a*) *Lymph-glands.*—The pharyngeal, thoracic, and abdominal groups are most frequently affected. The cervical, axillary, and inguinal are less commonly involved, but these glands may show slight enlargement. The lymphatic structures of the alimentary tract, the tissues of the tonsils, the adenoid structures in the upper pharynx, and the solitary and agminated follicles of the small and large intestines are usually much enlarged. The hyperplasia of the intestinal lymphatic structures may be the most remarkable, the individual glands standing out like peas.

(*b*) *Spleen.*—Enlargement of this organ is usually moderate in degree. The Malpighian bodies may show very prominently, and when anæmic may look like large tubercles. The organ is usually soft and hyperæmic.

(*c*) The *thymus* is enlarged, and may measure as much as 10 cm. in length. It looks swollen and soft, and on section may exude a milky white fluid.

(*d*) The *bone marrow* has been found in a state of hyperplasia, and the yellow marrow of the long bones in young adults, and even in persons between the ages of twenty and thirty, has been found replaced by red marrow. Among other associated conditions of this *constitutio lymphatica*, as it has been called, are hypoplasia of the heart and aorta and enlargement of the thyroid gland. In a large number of the cases in children rickets is coincident.

The *diagnosis* of the lymphatic constitution is not always easy. Enlargement of the superficial glands, with hypertrophy of the tonsils, signs of slight swelling of the thyroid, dulness over the sternum, with signs of enlargement of the mesenteric glands, are among the most important features. Signs of hypoplasia of the vascular system are still more uncertain, though Quincke believes that in such instances the left ventricle is dilated and the peripheral arteries may be much smaller than normal. The subjects are usually ill-developed and infantile in conformation.

Sudden Death in the status lymphaticus.—What has directed the attention of writers more particularly to this condition is the frequency with which it has been found in cases of unexpected death from very trifling and inadequate causes. A good deal of attention was directed to the subject by the death of the son of Professor Langhans, of Berlin, immediately after the preventive inoculation with the antitoxine of diphtheria. In another child death occurred under similar circumstances. The condition has also been met with in a number of cases of sudden death under anæsthetics, and I know of one instance during anæsthesia for adenoid

growths. Cases of sudden death of persons in the water, who have fallen in and, though immediately recovered, were dead, or who have died suddenly while bathing, are referred by Paltauf to this condition. And, lastly, there is the large group of cases of sudden death in children without recognizable cause, in whom post mortem the thymus has been found enlarged—the so-called "Thymus Tod" (see under Thymus Gland). It has also been suggested that certain of the sudden deaths during convalescence from the infectious fevers are to be referred to this status lymphaticus. Escherich thinks that certain measures usually harmless, such as hydrotherapy, may have an untoward effect in children in this condition of lymphatism, and adds that tetany and laryngismus may be associated with it.

The whole question is one which deserves the most careful study. The anatomical features appear fairly well defined. The clinical features are by no means so clear, nor is it at all certain in what way sudden death is caused in these cases. The students of the question have, however, in the past few years brought forward evidence enough to show that the subjects of this lymphatic constitution have a diminished vital resistance, and are especially prone to fatal collapse under ordinarily very inadequate exciting causes.

VIII. DISEASES OF THE SUPRARENAL BODIES.

1. Addison's Disease.

Definition.—A constitutional affection characterized by asthenia, depressed circulation, irritability of the stomach, and pigmentation of the skin. Tuberculosis of the adrenals is the common anatomical change. Recent observations indicate that the symptoms are due to loss of function of the suprarenal bodies.

The recognition of the disease is due to Addison, of Guy's Hospital, whose monograph on The Constitutional and Local Effects of Disease of the Suprarenal Capsules was published in 1855.

Etiology.—Males are more frequently attacked than females. In Greenhow's analysis of 183 cases 119 were males and 64 females. A majority of the cases occur between the twentieth and the fortieth year. A congenital case has been described in which the skin had a yellow-gray tint. The child lived for eight weeks, and post mortem the adrenals were found to be large and cystic. Injury such as a blow upon the abdomen or back, and caries of the spine, have in many cases preceded the attack. The disease is rare in America. The number of deaths during the census year 1890 was 99—59 males and 40 females. Twelve cases have come under my personal observation, 9 in men. One case was in a negro.

Morbid Anatomy and Pathology.—There is rarely emaciation or anæmia. Rolleston * thus summarizes the condition of the suprarenal bodies in Addison's disease:

* Gulstonian Lectures, Royal College of Physicians, British Medical Journal, 1895, i, to which the student is referred for an exhaustive consideration of the entire question.

"1. The fibro-caseous lesion due to tuberculosis—far the commonest condition found. 2. Simple atrophy. 3. Chronic interstitial inflammation leading to atrophy. 4. Malignant disease invading the capsules, including Addison's case of malignant nodule compressing the suprarenal vein. 5. Blood extravasated into the suprarenal bodies. 6. No lesion of the suprarenal bodies themselves, but pressure or inflammation involving the semilunar ganglia.

"The first is the only common cause of Addison's disease. The others, with the exception of simple atrophy, may be considered as very rare."

Among other anatomical features the condition of the abdominal sympathetic has been specially studied. The nerve-cells of the semilunar ganglia have been described as degenerated and deeply pigmented, and the nerves sclerotic. The ganglia are not uncommonly entangled in the cicatricial tissue about the adrenals. The spleen has occasionally been found enlarged; the thymus may have persisted and be larger than normal.

It is difficult to explain satisfactorily all the symptoms of this remarkable disease. The two chief theories which have been advanced are briefly as follows: (*a*) That the disease depended upon the loss of function of the adrenals. This was the view of Addison. The balance of experimental evidence is in favor of the view that the adrenals are functional glands, which furnish an internal secretion essential to the normal metabolism. Schäfer and Oliver have shown that the human adrenals contain a very powerful extract, which is not to be obtained in cases of Addison's disease; they have also studied the toxic effects on animals of the extracts of the glands. In the cases in which the adrenals have been found involved without the symptoms of Addison's disease, accessory glands may have been present; while in the rare cases in which the symptoms of the disease have been present with healthy adrenals the semilunar ganglia and adjacent tissues have been involved in dense adhesions, which may have interfered readily with the vessels or lymphatics of the glands. On this view Addison's disease is due to an inadequate supply of the adrenal secretion, just as myxœdema is caused by loss of function of the thyroid gland. "Whether the deficiency in this internal secretion leads to a toxic condition of the blood or to a general atony and apathy is a question which must remain open" (Rolleston). (*b*) That it is an affection of the abdominal sympathetic system, induced most commonly by disease of the adrenals, but also by other chronic disorders which involve the solar plexus and its ganglia. According to this view, it is an affection of the nervous system, and the pigmentation has its origin in changes induced through the trophic nerves. The pronounced debility is the outcome of disturbed tissue metabolism, and the circulatory, respiratory, and digestive symptoms are due to implication of the pneumogastric. The changes found in the abdominal sympathetic are held to support this view, and its advocates urge the occurrence of pigmentation of the skin in tuberculosis of the peritonæum, cancer of the pancreas, or aneurism of the abdominal aorta. Bramwell thinks that the symptoms may be in part due to irritation of the sympathetic and in part to renal inadequacy.

Symptoms.—In the words of Addison, the characteristic symptoms are "anæmia, general languor or debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin."

The onset is, as a rule, insidious. The feelings of weakness, as a rule, precede the pigmentation. In other instances the gastro-intestinal symptoms, the weakness, and the pigmentation come on together. There are a few cases in the literature in which the whole process has been acute, following a shock or some special depression. There are three important symptoms of the disease:

(1) *Pigmentation of the Skin.*—This, as a rule, first attracts the attention of the patient's friends. The grade of coloration ranges from a light yellow to a deep brown, or even black. In typical cases it is diffuse, but always deeper on the exposed parts and in the regions where the normal pigmentation is more intense, as the areolæ of the nipples and about the genitals; also wherever the skin is compressed or irritated, as by the waistband. At first it may be confined to the face and hands. Occasionally it is absent. Patches showing atrophy of pigment, leucoderma, may occur. The pigmentation is found on the mucous membranes of the mouth, conjunctivæ, and vagina. A patchy pigmentation of the serous membranes has often been found. Over the diffusely pigmented skin there may be little mole-like spots of deeper pigmentation. The pigmentation of the skin alone, unless the mucous membranes are also involved, is rarely sufficient in itself to make the diagnosis clear.

(2) *Gastro-intestinal Symptoms.*—The disease may set in with attacks of nausea and vomiting, spontaneous in character. Toward the close there may be pain with retraction of the abdomen, and even features suggestive of peritonitis (Ebstein). An intense anorexia may be present. The gastric symptoms are variable throughout the course; occasionally they are absent. Attacks of diarrhœa are frequent and come on without obvious cause.

(3) *Asthenia.*—This is perhaps the most characteristic feature of the disease. It may be manifested early as a feeling of inability to carry on the ordinary occupation, and the patient complains constantly of feeling tired. The weakness is specially marked in the muscular and cardio-vascular systems. There may be an extreme degree of muscular prostration in an individual apparently well nourished and whose muscles feel firm and hard. The cardio-vascular asthenia is manifest in a feeble, irregular action of the heart, which may come on in paroxysms, in attacks of vertigo, or of syncope, in one of which the disease may prove fatal. Headache is a frequent symptom; convulsions occasionally occur. McMunn has described an increase in the urinary pigments, and a pigment has been isolated of very much the same character as the melanin of the skin.

Anæmia was a symptom specially referred to by Addison, but it has been present in a marked degree in only one of my cases. I saw an instance, in Philadelphia, with J. C. Wilson, in which the diagnosis at first was not at all clear between Addison's disease and pernicious anæmia.

The mode of termination is either by syncope, which may occur even

early in the disease, by gradual progressive asthenia, or by the development of tuberculous lesions. In two cases I have known a noisy delirium with urgent dyspnœa to precede the fatal event.

Diagnosis.—Pigmentation of the skin is not confined to Addison's disease. The following are the conditions which may give rise to an increase in the pigment:

(1) Abdominal growths—tubercle, cancer, or lymphoma. In tuberculosis of the peritonæum pigmentation is not uncommon.

(2) Pregnancy, in which the discoloration is usually limited to the face, the so-called *masque des femmes ençeintes*. Uterine disease is a common cause of a patchy melasma.

(3) Hepatic disease, which may induce definite pigmentation, as in the diabetic cirrhosis. More commonly in overworked persons of constipated habit and with sluggish livers there is a patchy staining about the face and forehead.

(4) The vagabond's discoloration, caused by the irritation of lice and dirt, which may reach a very high grade, and has sometimes been mistaken for Addison's disease.

(5) In rare instances there is deep discoloration of the skin in melanotic cancer, so deep and general that it has been confounded with *melasma suprarenale*.

(6) In certain cases of exophthalmic goitre abnormal pigmentation occurs, as noted by Drummond and others.

(7) In a few rare instances the pigmentation common in scleroderma may be general and deep.

(8) In the face there may be an extraordinary degree of pigmentation due to innumerable small black comedones. If not seen in a very good light, the face may suggest argyria. Pigmentation of an advanced grade may occur in chronic ulcer of the stomach and in dilatation of the organ.

(9) Argyria could scarcely be mistaken, and yet I was consulted this year by a woman in whom the diagnosis of Addison's disease had been made by several good observers, but the character of the pigmentation, the length of time it had lasted, and her freedom from all symptoms pointed undoubtedly to argyria, though, so far as she or her physician knew, she had never taken nitrate of silver medicinally.

In any case of unusual pigmentation these various conditions must be sought for; the diagnosis of Addison's disease is scarcely justifiable without the asthenia. In many instances it is difficult early in the disease to arrive at a definite conclusion. The occurrence of fainting fits, of nausea, and gastric irritability are important indications. As the lesion of the capsules is almost always tuberculous, in doubtful cases the tuberculin test may be used. In a recent case, a robust, healthy-looking man with symptoms of Addison's disease, the characteristic reaction was obtained.

Prognosis.—The disease is usually fatal. The cases in which the bronzing is slight or does not occur run a more rapid course. There are occasionally acute cases which, with great weakness, vomiting, and diarrhœa, prove fatal in a few weeks. In a few cases the disease is much pro-

longed, even to six or ten years. In rare instances recovery has taken place, and periods of improvement, lasting many months, may occur.

Treatment.—The causal indications cannot be met. When there is profound asthenia the patient should be confined to bed, as fatal syncope may at any time occur. In three of my cases death was sudden. When anæmia is present iron may be given in full doses. Arsenic and strychnia are useful tonics. For the diarrhœa large doses of bismuth should be given; for the irritability of the stomach, creasote, hydrocyanic acid, ice, and champagne. The diet should be light and nutritious. Many patients thrive best on a strict milk diet.

Treatment by Suprarenal Extract.—Following the researches of Schäfer and Oliver, the latter used the gland in the treatment of the disease. Kinnicutt has collected 48 cases treated with adrenal preparations. Of these, 6 were reported as cured and 22 as improved. I have used it in 4 cases, of which one has been already reported. The patient was greatly benefited, gained 19 pounds, the symptoms of asthenia disappeared, and he was alive two years subsequently, but was still pigmented. The 3 other cases were not benefited in the slightest degree. The gland may be given raw or partially cooked or in a glycerin extract. Tabloids of the dried extract are used, one grain of which corresponds to fifteen of the gland. Three of the tabloids may be given daily. Operation has been suggested, but has not been carried out on any undoubted case.

2. Other Diseases of the Suprarenal Capsules.

Hæmorrhage into the gland is not uncommon, particularly in new-born children (Spencer). *Tuberculosis* may occur without the symptoms of Addison's disease. Among 157 cases of tuberculous disease in various parts of the body, caseous tuberculous foci were found in 20 in the suprarenals without signs of Addison's disease (Rolleston).

Tumors of the Suprarenals.—Adenomata are common, particularly the small yellowish nodules. *Fibromata* and *fatty* tumors occur, but are rare.

Of *malignant growths* secondary tumors are not uncommon. In 63 cases of secondary carcinoma, in 7 the suprarenal bodies were the seat of growths (Rolleston). Of the primary growths, both sarcoma and carcinoma may occur. Affleck and Leith have collected 20 cases of primary sarcoma. Ramsay informs me that we have had 3 cases of primary tumor of the suprarenals at the Johns Hopkins Hospital—2 in females and 1 in a male. Two were sarcomata and 1 a carcinoma. The diagnosis in all was malignant tumor of the kidney. The cases were operated upon, 1 with complete recovery.

IX. DISEASES OF THE SPLEEN.*

Apart from the acute swelling in fever, the chronic enlargement of the organ in paludism, leukæmia, cirrhosis of the liver, and heart-disease, we

* For a good discussion of the general pathology of the spleen, see Rolleston in Allbutt's System of Medicine.

see very few instances of disease of the spleen. These affections have been fully described, but there remain several conditions to which brief reference may be made.

1. Movable Spleen.

Movable or wandering spleen is seen most frequently in women the subjects of enteroptosis. It is occasionally met with without signs of displacement of other organs. It may be found accidentally in individuals who present no symptoms whatever. In other cases there are dragging, uneasy feelings in the back and side. All grades are met with, from a spleen that can be felt completely below the margin of the ribs to a condition in which the tumor is felt as low as the pelvis; indeed, the organ has been found in an inguinal hernia! In the large majority of all cases the spleen is enlarged. Sometimes it appears that the enlargement has caused relaxation of the ligaments; in other instances the relaxation seems congenital, as movable spleens have been found in different members of the same family. Possibly traumatism may account for some of the cases. Apart from the dragging, uneasy sensations and the worry in nervous patients, wandering spleen causes very few serious symptoms. Torsion of the pedicle may produce a very alarming and serious condition, leading to great swelling of the organ, high fever, or even to necrosis. A young woman was admitted to my colleague Kelly's ward with a tumor supposed to be ovarian, but which proved to be a wandering, moderately enlarged spleen. She was transferred to the medical ward, where she developed suddenly very great pain in the abdomen, a large swelling in the left flank, and much tenderness. Halsted operated and found an enormously enlarged spleen in a condition of necrosis, adherent to the adjacent parts and to the abdominal wall. He laid it open freely, and large necrotic masses of spleen tissue discharged for some time. She made a good recovery.

The *diagnosis* of a wandering spleen is usually easy unless the organ becomes fixed and is deformed by adhesions and perisplenitis. The shape of the organ and the sharp margin with the notches are the points to be specially noted.

The *treatment* of the condition is important. Occasionally the organ may be kept in position by a properly adapted belt and a pad under the left costal margin. Removal of the displaced organ has been advised and carried out in many cases, and nowadays it is not a very serious operation. It is, however, as a rule unnecessary. In 2 cases of enlarged spleen under my care, with great mobility, causing much discomfort and uneasiness, Halsted completely relieved the condition by replacing the spleen, packing it in position with gauze, and allowing firm adhesions to take place. Both these patients were seen more than eighteen months after the operation and the organ had remained in position.

2. Rupture of the Spleen.

This is of interest medically in connection with the spontaneous rupture in cases of acute enlargement during typhoid fever or malaria. The condition seems very rare in this country. We have had instances of rup-

ture of a malarial spleen following a blow, but neither in this disease nor in typhoid have we had an instance of spontaneous rupture. In India and in Mauritius rupture of the spleen is stated to be very common. Fatal hæmorrhage may follow puncture of a swollen spleen with a hypodermic needle. Occasionally the rupture results from the breaking of an infarct or of an abscess. The symptoms are those of hæmorrhage into the peritonæum, and the condition demands immediate laparotomy.

3. Infarct and Abscess of the Spleen.

Emboli in the splenic arteries causing infarcts may be either infective or simple. They are seen most frequently in ulcerative endocarditis and in septic conditions. Infarcts may also follow the formation of thrombi in the branches of the splenic artery in cases of fever. They are not very infrequent in typhoid. In a few instances the infarcts have followed thrombosis in the splenic veins. They are chiefly of pathological interest. The infarct of the spleen may be suspected in cases of septicæmia or pyæmia when there is pain in the splenic region, tenderness on pressure, and slight swelling of the organ; on several occasions I have heard a well-marked peritoneal friction rub. Occasionally in the infective infarcts large abscesses are formed, and in rare instances the whole organ may be converted into a sac of pus.

Tumors of the spleen, hydatid and other *cysts* of the organ, and *gummata* are rare conditions of anatomical interest, for an account of which the reader is referred to Rolleston's article and to the section on the spleen, by G. R. Lockwood, in Loomis and Thompson's System of Medicine.

4. Splenic Anæmia.

This condition, usually regarded as the splenic form of Hodgkin's disease, as such was well described, in 1871, by H. C. Wood. Strümpell, Banti, and others, however, think it should be separated and regarded as a special form. It is a disease characterized by great enlargement of the organ, profound anæmia, without leucocytosis and without the coexistence of malaria, rickets, or other states in which enlargement of the spleen is secondary; hence it is often spoken of as *primitive spleno-megaly.* While true primitive cases are rare, in this region, at least, an anæmia associated with enlargement of the spleen is not very uncommon, particularly as a result of the effects of prolonged residence in malarial regions. As I write a patient from South Carolina is in the wards with an enlarged spleen and great pallor. The anæmia is of a distinctly chlorotic type, as his blood-count is nearly 4,000,000, but the hæmoglobin is only 40 per cent. He has no leucocytosis. He has not had chills and fever for fifteen years, but has been living in a malarial region. There are cases, too, in which the enlarged spleen persists for many years with no anæmia, good color, and a fair muscular vigor. I remember a soldier invalided from India, admitted to the Montreal General Hospital with an enormous spleen and slight anæmia. He died shortly after admission of a profuse hæmorrhage from

the stomach. A patient from Jamaica, referred to me a few years ago by Henderson, of Kingston, without any malarial history, had an enormous spleen, had had several attacks of profound anæmia, but at the time of observation had a blood-count nearly normal. I see many more cases of primitive spleno-megaly without than with anæmia.

S. West, in Allbutt's System, gives the following as the main features of splenic anæmia: "The disease may be divided into three stages: In the initial stage the symptoms are those of extreme anæmia, with great loss of muscular power and some wasting of muscle, though usually without emaciation. As in this stage the disease presents no specific features, it can rarely be recognized. The second stage is characterized by progressive enlargement of the spleen and by attacks of severe pain in the splenic region; the anæmia is more profound, the loss of strength is extreme, and the patients are liable to repeated attacks of bleeding, especially from the nose; the temperature is now usually raised and of hectic character, reaching 102° or more in the evening. It is in this second stage that the disease is first recognized.

"In the last stage the condition is one of progressive asthenia, which ends in death; there is in it nothing especially characteristic."

The blood condition is one simply of profound anæmia without increase in the leucocytes and not always with marked poikilocytosis. The tendency to hæmorrhage is marked, both from the mucous surfaces and in the skin.

Anatomically, the only special changes that have been noted have been a peculiar atrophy of the Malpighian corpuscles in some cases.

The treatment of the condition is that of other forms of profound anæmia.

X. DISEASES OF THE THYROID GLAND.

1. Goitre.

Definition.—Hypertrophy of the thyroid gland, occurring sporadically or endemically.

In this country sporadic cases are common. The endemic centres referred to in Barton's monograph (1810) and in Hirsch's Geographical Pathology no longer exist. The disease is very prevalent about the eastern end of Lake Ontario, and in parts of Michigan (Dock). Endemically it is found particularly in the mountainous regions of Switzerland and in parts of Italy. No satisfactory explanation has been given of the existence of the disease in this form.

Anatomically the following varieties may be distinguished: (*a*) Parenchymatous, in which the enlargement is general and the follicles, usually newly formed, contain a gelatinous colloid material. (*b*) Vascular, in which the enlargement is chiefly due to dilatation of the blood-vessels without the new formation of glandular tissue. (*c*) Cystic goitre, in which the enlarged gland is occupied by large cysts, the walls of which often undergo calcification.

Symptoms.—The enlargement may be uniform throughout the entire gland, or affect only one lobe, or the isthmus alone. When small, a goitre causes no inconvenience. In its growth it may compress the trachea, causing dyspnœa, or may pass beneath the sternum and compress the veins. These, however, are exceptional circumstances, and in a large proportion of all cases no serious symptoms are noted. The affection usually comes under the care of the surgeon. Sudden death occasionally occurs in large bronchoceles. In some instances it may be difficult to determine the cause, and it has been thought to be associated with pressure on the vagi. I have reported an instance in which it resulted from hæmorrhage into the gland and into the adjacent tissues. The blood passed into the cellular tissues of the neck and under the sternum, covering the aorta and pericardium. In regions in which goitre prevails the drinking-water should be boiled. Change of locality is sometimes followed by cure. The medicinal treatment is very unsatisfactory. Iodine and various counterirritants externally, iodide of potash, ergot, and many other drugs are recommended by writers. The thyroid extract has been used with success by Bruns in 9 of 12 cases.

2. Tumors of the Thyroid.

These are very varied. (*a*) Adenomata, either simple or malignant. The latter may form extensive metastases. A case is reported by Hayward in which growths resembling thyroid tissue occurred in the lungs and various bones of the body. (*b*) Cancer, of which several forms have been described. (*c*) Sarcoma. All of these have a surgical rather than a medical interest.

It may be mentioned that the aberrant or accessory thyroid gland may form large tumors in the mediastinum or in the pleura. Cases have been reported by F. A. Packard and myself, and an instance is on record in which an enormous cystic accessory thyroid occupied the entire right pleura.

Lingual goitre occasionally develops at the base of the tongue, and is an enlarged accessory thyroid in that situation. It may lead to difficult deglutition and interference with articulation.

Thyroid abscess is rare. In Havel's monograph on Strumitis (1892) cases are given after nearly every one of the specific diseases, and he reports 18 cases from Kocher's clinic, nearly all secondary or metastatic.

3. Exophthalmic Goitre (*Parry's Disease*).

Definition.—A disease characterized by exophthalmos, enlargement of the thyroid, and functional disturbance of the vascular system. It is very possibly caused by disturbed function of the thyroid gland (hyperthyroidism).

Historical Note.—In the posthumous writings of Caleb Hillier Parry (1825) is a description of 8 cases of Enlargement of the Thyroid Gland in Connection with Enlargement or Palpitation of the Heart. In the first case, seen in 1786, he also describes the exophthalmos: "The eyes were pro-

Dr. H. S. Muir of Truro - N. S. tells me that the disease is very common in that province.

Among the symptoms which Madame T. (our Batesboro's second patient) presented was that of being unable to keep her "hands quiet."
He mentions this later on in his history of the case in the following words: "ruhelose Haltung ihres Körpers und der Hände"

Walter R. Steiner

truded from their sockets, and the countenance exhibited an appearance of agitation and distress, especially in any muscular movement." The Italians claim that Flajani described the disease in 1800. I have not been able to see his original account, but Moebius states that it is meagre and inaccurate, and bears no comparison with that of Parry. If the name of any physician is to be associated with the disease, undoubtedly it should be that of the distinguished old Bath physician. Graves described the disease in 1835 and Basedow in 1840.

Etiology.—The disease is more frequent in women than in men. Of 200 cases tabulated by Eshner, there were 161 females. The age of onset is usually from the twentieth to the thirtieth year. It is sometimes seen in several members of the same family. Worry, fright, and depressing emotions precede the development of the disease in a number of cases.

The disease is regarded by some as a pure neurosis, in favor of which is urged the onset after a profound emotion, the absence of lesions, and the cure which has followed in a few cases after operations upon the nose. Others believe that it is caused by a central lesion in the medulla oblongata. In support of this there is a certain amount of experimental evidence, and in a few autopsies changes have been found in the medulla. Of late years the view has been urged, particularly by Moebius and by Greenfield, that exophthalmic goitre is primarily a disease of the thyroid gland (*hyperthyrea*), in antithesis to myxœdema (*athyrea*). The clinical contrast between these two diseases is most suggestive—the increased excitability of the nervous system, the flushed, moist skin, the vascular erythism in the one; the dull apathy, the low temperature, slow pulse, and dry skin of the other. The changes in the gland in exophthalmic goitre are, as shown by Greenfield, those of an organ in active evolution—viz., increased proliferation, with the production of newly formed tubular spaces and absorption of the colloid material which is replaced by a more mucinous fluid (Bradshaw Lecture, 1893). The thyroid extract given in excess produces symptoms not unlike those of Parry's disease—tachycardia, tremor, headache, sweating, and prostration. Beclère has recently reported a case in which exophthalmos developed after an overdose. Use of the thyroid extract usually aggravates the symptoms of exophthalmic goitre. The most successful line of treatment has been that directed to diminish the bulk of the goitre. These are some of the considerations which favor the view that the symptoms are due to disturbed function of the thyroid gland, probably to a hypersecretion of certain materials, which induce a sort of chronic intoxication. Myxœdema may develop in the late stages, and there are transient œdema and in a few cases scleroderma, which indicate that the nutrition of the skin is involved. Persistence of the thymus is almost the rule (Hector Mackenzie), but its significance is unknown.

Symptoms.—Acute and chronic forms may be recognized. In the acute form the disease may develop with great rapidity. In a patient of J. H. Lloyd's, of Philadelphia, a woman, aged thirty-nine, who had been considered perfectly healthy, but whose friends had noticed that for some time her eyes looked rather large, was suddenly seized with intense vomiting and diarrhœa, rapid action of the heart, and great throbbing of the

arteries. The eyes were prominent and staring and the thyroid gland was found much enlarged and soft. The gastro-intestinal symptoms continued, the pulse became more rapid, the vomiting was incessant, and the patient died on the third day of the illness. Only the abdominal and thoracic organs could be examined and no changes were found. Two rapidly fatal cases occurred at the Philadelphia Hospital, one of which, under F. P. Henry's care, had marked cerebral symptoms. The acute cases are not always associated with delirium. In a case reported by Sutcliff death occurred within three months from the onset of the symptoms, owing to repeated and uncontrollable vomiting. More frequently the onset is gradual and the disease is chronic. There are four characteristic symptoms of the disease—exophthalmos, tachycardia, enlargement of the thyroid, and tremor.

Tachycardia.—Rapid heart action is only one of a series of remarkable vascular phenomena in the disease. The pulse-rate at first may be not more than 95 or 100, but when the disease is established it may be from 140 to 160, or even higher. Irregularity is not common, except toward the close. In a well-developed case the visible area of cardiac pulsation is much increased, the action is heaving and forcible, and the shock of the heart-sounds is well felt. The large arteries at the root of the neck throb forcibly. There is visible pulsation in the peripheral arteries. The capillary pulse is readily seen, and there are few diseases in which one may see at times with greater distinctness the venous pulse in the veins of the hand. The throbbing pulsation of the arteries may be felt even in the finger tips. On auscultation murmurs are usually heard over the heart, a loud apex systolic and loud bruits at the base and over the manubrium. The sounds of the heart may be very intense. In rare instances they may be heard at some distance from the patient; according to Graves, as far as four feet.

Exophthalmos, which may be unilateral, usually follows the vascular disturbance. It is readily recognized by the protrusion of the balls, and partly by the fact that the lids do not completely cover the sclerotics, so that a rim of white is seen above and below the cornea. The protrusion may become very great and the eye may even be dislocated from the socket, or both eyes may be destroyed by panophthalmitis, a condition present in one of Basedow's cases. The vision is normal. Graefe noted that when the eyeball is moved downward the upper lid does not follow it as in health. This is known as Graefe's sign. It seems to be rare; it was not present in any one of 17 cases examined at my clinic (Oppenheimer). The palpebral aperture is wider than in health, owing to spasm or retraction of the upper lid (Stellwag's sign). The patient winks less frequently than in health. Moebius has called attention to the lack of convergence of the two eyes. Changes in the pupils and in the optic nerves are rare. Pulsation of the retinal arteries is common.

Enlargement of the thyroid commonly develops with the exophthalmos. It may be general or in only one lobe, and is rarely so large as in ordinary goitre. The vessels are usually much dilated, and the whole gland may be seen to pulsate. A thrill may be felt on palpation and on auscultation

a loud systolic murmur, or more commonly a *bruit de diable.* A double murmur is common and is pathognomonic (Guttmann).

Tremor is the fourth cardinal symptom, and was really first described by Basedow. It is involuntary, fine, about eight to the second. It is of great importance in the diagnosis of the early cases.

Among other symptoms which may develop are anæmia, emaciation, and slight fever. Attacks of vomiting and diarrhœa may occur. The latter may be very severe and distressing, recurring at intervals. The greatest complaint is of the forcible throbbing in the arteries, often accompanied with unpleasant flushes of heat and profuse perspirations. Skin symptoms are not infrequent—pigmentation, which may be intense and simulate Addison's disease, patches of leucoderma, or atrophy of pigment, and urticaria. Patches of solid œdema have been seen. Occasionally myxœdema has been present. In the very acute case above referred to urticaria was a prominent symptom. Occasionally pruritus is an early and most distressing symptom. I have seen one case in which it persisted and became almost unbearable. Irritability of temper, change in disposition, and great mental depression have been described. An important complication is acute mania, in which the patient may die in a few days. Weakness of the muscles is not uncommon, particularly a feeling of "giving way" of the legs. If the patient holds the head down and is asked to look up without raising the head, the forehead remains smooth and is not wrinkled, as in a normal individual (Joffroy). A feature of interest noted by Charcot is the great diminution in the electrical resistance, which may be due to the saturation of the skin with moisture owing to the vaso-motor dilatation (Hirt). Bryson has noted the fact that the chest expansion may be greatly diminished. The emaciation may be extreme. Glycosuria and albuminuria are not infrequent complications. True diabetes may also develop.

The course of the disease is usually chronic, lasting several years. After persisting for six months or a year the symptoms may disappear. There are remarkable instances in which the symptoms have come on with great intensity, following fright, and have disappeared again in a few days. A certain proportion of the cases get well, but when the disease is well developed recovery is rare.

Treatment.—Medicinal measures are notoriously uncertain. The combination of digitalis and iron may be tried, and, when there is anæmia, often does good. I have never seen any advantage from the use of aconite or veratrum viride. The tincture of strophanthus will sometimes reduce the rapidity of the heart's action. Ergot is warmly recommended by some writers. Belladonna gives relief occasionally, and should be administered until the dryness of the throat is obtained. I have seen one case of apparent cure under its use. No measures are so successful as rest in bed with an ice-bag or Leiter's tube applied occasionally over the heart, or, what is sometimes more agreeable, over the lower part of the neck and manubrium sterni. I have known the pulse to be reduced in this way from 140 to 90. Electricity has been much lauded and instances of cure have been reported. In many cases temporary improvement certainly follows the use of the galvanic current. Erb states that the anode should

be placed over the cervical spine and the cathode upon the peripheral nerves. The use of the thyroid extract has not been successful. The thymus extract has not proved satisfactory. The treatment of the disease by small doses of opium has been successful in some cases (Musser). Operative measures seem to offer the greatest relief. Removal of one lobe of the gland, tying the arteries of the gland, and exothyropexia have all been tried. The patients, as a rule, stand the anæsthetic badly; death on the table is more frequent than the published records indicate. Recently good results have been reported from the division of the cords of the cervical sympathetic.

4. Myxœdema (*Athyrea*).

Definition.—A constitutional affection, due to the loss of function of the thyroid gland. The disease, which was described by Sir William Gull as a cretinoid change, and later by Ord, is characterized clinically by a myxœdematous condition of the subcutaneous tissues and mental failure, and anatomically by atrophy of the thyroid gland.

Clinical Forms.—Three groups of cases may be recognized—cretinism, myxœdema proper, and operative myxœdema.

Cretinism.

This remarkable impairment of nutrition follows absence or loss of function of the thyroid gland, either congenital or appearing at any time before puberty. There is remarkable retardation of development, retention of the infantile state, and an extraordinary disproportion between the different parts of the body. Two forms of cretinism are recognized, the *sporadic* and the *endemic*. In the sporadic form the gland may be congenitally absent, it may be atrophied after one of the specific fevers, or the condition may develop with goitre. Since we have learned to recognize the disease it is surprising how many cases have been reported. I was able to collect 60 cases in this country to May 1, 1897.*

The condition is rarely recognized before the infant is six or seven months old. Then it is noticed that the child does not grow so rapidly and is not bright mentally. The tongue looks large and hangs out of the mouth. The hair may be thin and the skin very dry. Usually by the end of the first year and during the second year the signs of cretinism become very marked. The face is large, looks bloated, the eyelids are puffy and swollen; the alæ nasi are thick, the nose looks depressed and flat. Dentition is delayed, and the teeth which appear decay early. The abdomen is swollen, the legs are thick and short, and the hands and feet are undeveloped and pudgy. The face is pale and sometimes has a waxy, sallow tint. The fontanelles remain open; there is much muscular weakness, and the child cannot support itself. In the supraclavicular regions there are large pads of fat. The child does not develop mentally; there are various grades of idiocy and imbecility.

* Sporadic cretinism in America, Transactions of the Congress of American Physicians and Surgeons, vol. iv.

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Jones cho. claims remarkable results in fifteen cases.

A very interesting form is that in which, after the child has thriven and developed until its fourth or fifth year, or even later, the symptoms begin after a fever, in consequence of an atrophy of the gland. Parker suggests for this variety the name juvenile myxœdema.

Endemic cretinism develops under local conditions, as yet unknown, in association with goitre. It is met with chiefly in Switzerland and parts of Italy and France. The common opinion is that it too is associated with loss of function of the thyroid.

The *diagnosis* of cretinism is very easy after one has seen a case or good illustrations. Infants a year or so old sometimes become flabby, lose their vivacity, or show a protuberant abdomen and lax skin with slight cretinoid appearance. These milder forms, as they have been termed, are probably due to transient functional disturbance in the gland. There is rarely any difficulty in recognizing the different other types of idiocy. The condition known as *fœtal rickets, achondroplasia,* or the *chondrodystrophia fœtalis,* is more likely to be mistaken for cretinism. The children which survive birth grow up as a remarkable form of dwarfs, characterized by shortness of the limbs (micromelia) and enormous enlargement of the articulations, due to hyperplasia of the cartilaginous ends of the bones. *Infantilism*—the condition characterized by a preservation in the adult of the exterior form of infancy with the non-appearance of the secondary sexual characters—could scarcely be mistaken for cretinism.

Myxœdema of Adults (*Gull's Disease*).

In this, women are very much more frequently affected than men—in a ratio of 6 to 1. The disease may affect several members of a family, and it may be transmitted through the mother. In some instances there has been first the appearance of exophthalmic goitre. Though occurring most commonly in women, it seems to have no special relation to the catamenia or to pregnancy; the symptoms of myxœdema may disappear during pregnacy or may develop post partum. Myxœdema and exophthalmic goitre may occur in sisters. It is not so common in this country as in England. The symptoms of this form, as given by Ord,* are marked increase in the general bulk of the body, a firm, inelastic swelling of the skin, which does not pit on pressure; dryness and roughness, which tend, with the swelling, to obliterate in the face the lines of expression; imperfect nutrition of the hair; local tumefaction of the skin and subcutaneous tissues, particularly in the supraclavicular region. The physiognomy is altered in a remarkable way: the features are coarse and broad, the lips thick, the nostrils broad and thick, and the mouth is enlarged. Over the cheeks, sometimes the nose, there is a reddish patch. There is a striking slowness of thought and of movement. The memory becomes defective, the patients grow irritable and suspicious, and there may be headache. In some instances there are delusions and hallucinations, leading to a final condition of dementia. The gait is heavy and slow. The temperature may be below

* Report on Myxœdema, Clinical Society's Transactions, 1888.

normal. The functions of the heart, lungs, and abdominal organs are normal. Hæmorrhage sometimes occurs. Albuminuria is sometimes present, more rarely glycosuria. Death is usually due to some intercurrent disease, most frequently tuberculosis (Greenfield). The thyroid gland is diminished in size and may become completely atrophied and converted into a fibrous mass. The subcutaneous fat is abundant, and in one or two instances a great increase in the mucin has been found.

The course of the disease is slow but progressive, and extends over ten or fifteen years. A condition of acute and temporary myxœdema may develop in connection with enlargement of the thyroid in young persons. Myxœdema may follow exophthalmic goitre. In other instances the symptoms of the two diseases have been combined. I have reported a case in which a young man became bloated and increased in weight enormously during three months, then developed tachycardia with tremor and active delirium, and died within six months of the onset of the symptoms.

Operative Myxœdema; Cachexia Strumipriva.

Horsley, in a series of interesting experiments, showed that complete removal of the thyroid in monkeys was followed by the production of a condition similar to that of myxœdema and often associated with spasms or tetanoid contractures, and followed by apathy and coma. When the monkeys were kept warm myxœdema was averted, and, instead of an acute myxœdema, the animals developed a condition which closely resembled cretinism. An identical condition may follow extirpation of the thyroid in man. Kocher, of Bern, found that after complete extirpation a cachectic condition followed in many cases, the symptoms of which are practically identical with those of myxœdema. The disease follows only a certain number of total and a much smaller proportion of partial removals of the thyroid gland. Of 408 cases, in 69 the operative myxœdema developed. It has been thought that if a small fragment of the thyroid remains, or if there are accessory glands, which in animals are very common, these symptoms do not develop. It is possible that in men, in the cases of complete removal, the accessory fragments subserve the function of the gland. Operative myxœdema is very rare in America; I have been able to find only 2 cases in this country. McGraw's case, referred to in previous editions of this work, has since been cured with the thyroid extract.

The *diagnosis* of myxœdema is easy, as a rule. The general aspect of the patient—the subcutaneous swelling and the pallor—suggests Bright's disease, which may be strengthened by the discovery of tube-casts and of albumin in the urine; but the solid character of the swelling, the exceeding dryness of the skin, the yellowish-white color, the low temperature, the loss of hair, and the dull, listless mental state should suffice to differentiate the two conditions. In dubious cases not too much stress should be laid upon the supraclavicular swellings. There may be marked fibro-fatty enlargements in this situation in healthy persons, the supraclavicular pseudo-lipomata of Verneuil.

Treatment.—The patients suffer in cold and improve greatly in warm weather. They should therefore be kept at an even temperature, and should, if possible, move to a warm climate during the winter months. Repeated warm baths with shampooing are useful. Our art has made no more brilliant advance than in the cure of these disorders due to disturbed function of the thyroid gland. That we can to-day rescue children otherwise doomed to helpless idiocy—that we can restore to life the hopeless victims of myxœdema—is a triumph of experimental medicine for which we are indebted very largely to Victor Horsley and to his pupil Murray. Transplantation of the gland was first tried; then Murray used an extract subcutaneously. Hector Mackenzie in London and Howitz in Copenhagen introduced the method of feeding. We now know that the gland, taken either fresh, or as the watery or glycerin extract, or dried and powdered, is equally efficacious in a majority of all the cases of myxœdema in infants or adults. Many preparations are now on the market, but it makes little difference how the gland is administered. The dried powdered gland and the glycerin extract are most convenient. It is well to begin with the powdered gland, 1 grain three times a day, of the Parke-Davis preparation, or one of the Burroughs and Welcome tablets. The dose may be increased gradually until the patient takes 10 or 15 grains in the day. In many cases there are no unpleasant symptoms; in others there are irritation of the skin, restlessness, rapid pulse, and delirium; in rare instances tonic spasms, the condition to which the term *thyroidism* is applied. The results, as a rule, are most astounding—unparalleled by anything in the whole range of curative measures. Within six weeks a poor, feeble-minded, toad-like caricature of humanity may be restored to mental and bodily health. Loss of weight is one of the first and most striking effects; one of my patients lost over 30 pounds within six weeks. The skin becomes moist, the urine is increased, the perspiration returns, the temperature rises, the pulse-rate quickens and the mental torpor lessens. Ill effects are rare. Two or three cases with old heart lesions have died during or after the treatment; in one instance a temporary condition of Graves' disease was induced.

The treatment, as Murray suggests, must be carried out in two stages—one, early, in which full doses are given until the cure is effected; the other, the permanent use of small doses sufficient to preserve the normal metabolism. The literature of thyroid therapy and a list of all the cases of myxœdema and cretinism treated to December 31, 1894, are given by Heinsheimer.*

XI. DISEASES OF THE THYMUS GLAND.

The functions of this gland are unknown. It is a suggestive fact that Baumann found in it minute quantities of a compound containing iodine. It has been thought that its internal secretion has an influence in combating infective agents. The weight of the organ is about 14 grammes at birth, about 20 at the ninth month, and 25 to 30 at the second year.

* Die Schilddrüsenbehandlung, München, 1895.

The organ, after reaching its largest size about the end of the second year, gradually wastes, until at the time of puberty it is a mere fatty remnant, in which, however, there are "traces of its original structure in the form of small masses of thymus corpuscles, and even of concentric corpuscles" (Quain). A complete consideration of the affections of this gland is to be found in Friedleben's remarkable monograph, Die Physiologie der Thymusdrüse, 1858. The following are the most important conditions:

I. **Persistence of the organ** after the fifteenth year, met with occasionally, but under circumstances so varied that a satisfactory explanation cannot be offered. It is said that the existence of the gland may be determined by the presence of an area of dulness along the left sternal border from the second to the fourth ribs.

II. **Hypertrophy of the Thymus.**—The size of the gland varies widely, so that it is difficult to define exactly the limits between persistence and enlargement. The condition is of interest from three standpoints: (*a*) The supposed occurrence of *thymic asthma*, due to pressure from the enlarged gland. A number of observers have attributed the symptoms of laryngismus stridulus to pressure exerted by the enlarged thymus. Many German writers consider thymic asthma identical with the laryngismus stridulus of English authors, who, as a rule, have laid no stress whatever on the association. There can be, I think, no question that the ordinary laryngismus seen in rickety children is a convulsive affection and is not the result of compression. But a very greatly enlarged thymus may seriously hamper the structures within the thorax. Jacobi, in his monograph on the gland (Transactions of the Association of American Physicians, vol. iii), states that in an infant of eight months the distance between the manubrium sterni and the vertebral column is 22 cm., a space which he thinks might be completely filled by an enlarged and congested thymus. Siegel's case also points to the possibility of this compression. A boy aged two years and a half had had for two weeks cough and bronchial rales with dyspnœa, which was more or less constant with nocturnal exacerbations. Laryngismus stridulus was diagnosed. Tracheotomy was performed shortly after admission without relief, but when subsequently the anterior mediastinum was opened from above by extending the incision from the tracheotomy wound, a piece of the thymus as large as a hazel-nut appeared with each inspiration. The gland was drawn up with forceps and fastened by three stitches to the fascia over the sternum. The child rested quietly after the operation, had no dyspnœa, and made a complete recovery (Berl. klin. Woch., 1896, No. 40). From a child aged two months (dyspnœic from the eighth day) Koenig removed a portion of the thymus, leaving the substernal part. These are cases that go far to disprove Friedleben's dictum—*es giebt kein asthma thymicum.*

(*b*) *Thymus Enlargement and Sudden Death.*—In considering the question of the so-called lymphatic constitution, with which an enlarged thymus is usually associated, we have spoken of the occurrence of sudden death. Two groups of cases are met with in the literature: First, such instances as those described by Grawitz, Jacobi, and others, in which young infants have been either found dead in bed or have been attacked suddenly with

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dyspnœa, have become cyanotic and died in a few minutes. In such cases the thymus has been found greatly enlarged, and death has been thought to be directly due either to pressure on the air-passages, pressure on the pneumogastric (causing spasm of the glottis), or pressure on the great vessels. To the second group belong the cases in adults which have been described of late by Nordmann, Paltauf, Ohlmacher, and others, in which the sudden death has occurred under such conditions as anæsthesia or while bathing. In a number of these cases not only has the thymus been found enlarged, but the spleen and lymphatic tissues generally. The question is one of considerable medico-legal interest, and has been spoken of under Lymphatism.

Rolleston reports a case of sudden death after signs of cardiac failure lasting for only twenty minutes, in which there was hyperplasia of a persistent thymus. The gland with the trachea weighed 11 ounces.

(*c*) *Thymus Gland and Exophthalmic Goitre.*—That there is some association between these conditions is urged on two grounds: First, the persistence of the gland in Graves' disease. W. W. Ord and Hector Mackenzie state that it has been found enlarged in all the cases recently examined at St. Thomas's Hospital. Hektoen concludes from a very thorough study of the question that the coexistence is more than accidental. Secondly, the good results which are stated to follow the feeding of the thymus gland in Graves' disease are held to bear out the idea that the enlargement during life is compensatory. The general conclusion, however, reached by Hector Mackenzie and by Kinnicutt is that the thymus feeding has at best only slight influence upon Graves' disease.

It is interesting to note in connection with the question of enlarged thymus and sudden death that two of Hale White's cases of exophthalmic goitre died suddenly, and autopsy showed no reasonable cause of death.

Among other conditions with which enlarged thymus has been associated may be mentioned epilepsy (Ohlmacher).

III. **Other Morbid Conditions of the Thymus.**—*Hæmorrhages* are not uncommon, and are found particularly in children who have died of asphyxia.

Tumors of the gland, particularly sarcoma and lympho-sarcoma, have been frequently described. Many mediastinal tumors originate in the remnants of the thymus. Dermoid tumors and cysts have also been met with. Tuberculosis of the gland, chiefly in the form of miliary nodules, is well described in Jacobi's monograph. There is a well-authenticated case in which it was primary. Focal necroses in diphtheria have also been described by Jacobi.

Abscess of the Thymus.—Dubois, in 1850, noted the occurrence of foci of suppuration in the gland in subjects of congenital syphilis. Throughout it round or fissure-like cavities are seen filled with a purulent fluid. Chiari states that some of these supposed abscesses are areas of post-mortem softening, or cysts lined with flattened epithelium containing detritus of thymus cells. In one case Jacobi found a small gumma.

SECTION IX.

DISEASES OF THE KIDNEYS.

I. MALFORMATIONS.

Newman classifies the malformations of the kidney as follows: A. Displacements without mobility—(1) congenital displacement without deformity; (2) congenital displacement with deformity; (3) acquired displacements. B. Malformations of the kidney. I. Variations in number—(*a*) supernumerary kidney; (*b*) single kidney, congenital absence of one kidney, atrophy of one kidney; (*c*) absence of both kidneys. II. Variations in form and size—(*a*) general variations in form, lobulation, etc.; (*b*) hypertrophy of one kidney; (*c*) fusion of two kidneys—horseshoe kidney, sigmoid kidney, disk-shaped kidney. C. Variations in pelvis, ureters, and blood-vessels.

The fused kidneys may form a large mass, which is often displaced, being either in an iliac fossa or in the middle line of the abdomen, or even in the pelvis. Under these circumstances it may be mistaken for a new growth. In Polk's case the organ was removed under the belief that it was a floating kidney.* The patient lived eleven days, had complete anuria, and it was found post mortem that a single unsymmetrical kidney, as this form is called, had been removed.

II. MOVABLE KIDNEY.

(*Floating Kidney; Palpable Kidney; Ren mobilis; Nephroptosis*).

The kidney is held in position by its fatty capsule, by the peritonæum which passes in front of it, and by the blood-vessels. Normally the kidney is firmly fixed, but under certain circumstances one or another organ, more rarely both, becomes movable. In very rare cases the kidney is surrounded, to a greater or less extent, by the peritonæum, and is anchored at the hilus by a mesonephron. Some would limit the term floating kidney to this condition.

Movable kidney is almost always acquired. It is more common in

* New York Medical Journal, 1883.

women. Of the 667 cases collected in the literature by Kuttner, 584 were in women and only 83 in men. It is more common on the right than on the left side. Of 727 cases analyzed by this author, it occurred on the right in 553 cases, on the left in 81, and on both sides in 93. The greater frequency of the condition in women may be attributed to compression of the lower thoracic zone by tight lacing, and, more important still, to the relaxation of the abdominal walls which follows repeated pregnancies. This does not account for all the cases, as movable kidney is by no means uncommon in nulliparæ. Drummond believes that in a majority of the cases there is a congenitally relaxed condition of the peritoneal attachments. The condition has been met with in infants. Wasting of the fat about the kidney may be a cause in some instances. Trauma and the lifting of heavy weights are occasionally factors in its production. The kidney is sometimes dragged down by tumors. The greater frequency on the right side is probably associated with the position of the kidney just beneath the liver, and the depression to which the organ is subjected with each descent of the diaphragm in inspiration.

And, lastly, movable kidney is met with in many cases which present that combination of neurasthenia with gastro-intestinal disturbance which has been described by Glénard as *enteroptosis* (see p. 541).

To determine the presence of a movable kidney the patient should be placed in the dorsal position, with the head moderately low and the abdominal walls relaxed. The left hand is placed in the lumbar region behind the eleventh and twelfth ribs; the right hand in the hypochondriac region, in the nipple line, just under the edge of the liver. Bimanual palpation may detect the presence of a firm, rounded body just below the edge of the ribs. If nothing can be felt, the patient should be asked to draw a deep breath, when, if the organ is palpable, it is touched by the fingers of the right hand. Various grades of mobility may be recognized. It may be possible barely to feel the lower edge on deep palpation—*palpable kidney*—or the organ may be so far displaced that on drawing the deepest breath the fingers of the right hand may be, in a thin person, slipped above the upper end of the organ, which can be readily held down, but cannot be pushed below the level of the navel—*movable kidney*. In a third group of cases the organ is freely movable, and may even be felt just above Poupart's ligament, or may be in the middle line of the abdomen, or can even be pushed over beyond this point. To this the term *floating kidney* is appropriate.

The movable kidney is not painful on pressure, except when it is grasped very firmly, when there is a dull pain, or sometimes a sickening sensation. Examination of the patient from behind may show a distinct flattening in the lumbar region on the side in which the kidney is mobile.

Symptoms.—In a large majority of cases there are no symptoms, and if detected accidentally it is well not to let the patient know of its presence. Far too much stress has been laid upon the condition of late years. In other instances there is pain in the lumbar region or a sense of dragging and discomfort, or there may be intercostal neuralgia. In a large group the symptoms are those of neurasthenia with dyspeptic disturbance. In

women the hysterical symptoms may be marked, and in men various grades of hypochondriasis. The gastric disturbance is usually a form of nervous dyspepsia. Dilatation of the stomach has been observed, owing, as suggested by Bartels, to pressure of the dislocated kidney upon the duodenum. This view has been supported by Oser, Landau, and Ewald. On the other hand, Litten holds that the dilatation of the stomach is the cause of the mobility of the kidney, and he found in 10 cases of depression and dilatation of the stomach 22 instances of dislocation of the kidney on the right side. My own experience coincides with that of Drummond, who has very exceptionally found the two conditions to coexist. The association, however, with a *depressed* stomach is certainly not uncommon in women. Constipation is not infrequent. Some writers have described pressure upon the gall-ducts, with jaundice, but it is not very likely to occur. Fæcal accumulation and even obstruction may be associated with the displaced organ.

Dietl's Crises.—In floating kidney there are attacks characterized by severe abdominal pain, chills, nausea, vomiting, fever, and collapse. Scarcely any mention is made of such symptoms, which were first described by Dietl in 1864, and a more widespread knowledge of their occurrence in connection with this condition is desirable. My attention was called to them in 1880 by Palmer Howard in the case of a stout lady, who suffered repeatedly with the most severe attacks of abdominal pain and vomiting, which constantly required morphia. A tumor was discovered a little to the right of the navel, and the diagnosis of probable neoplasm was concurred in by Flint (Sr.) and Gaillard Thomas. The patient lost weight rapidly, became emaciated, and in the spring of 1881 again went to New York, where she saw Van Buren, who diagnosed a floating kidney and said that these paroxysms were associated with it in a gouty person. He cut off all stimulants, reassured the lady that she had no cancer, and from that time she rapidly recovered, and the attacks have been few and far between. In this patient any overindulgence in eating or in drinking is still liable to be followed by a very severe attack. These attacks may also be mistaken for renal colic, and the operation of nephrotomy has been performed.

In other instances the attacks of pain may be thought to be due to intestinal disease or to recurring appendicitis. The cause of these paroxysmal attacks is not quite clear. Dietl thought they were due to strangulation of the kidney or to twists or kinks in the renal vessels due to the extreme mobility. During the attacks the urine is sometimes high-colored and contains an excess of uric acid or of the oxalates. It is stated, too, that blood or pus may be present. The kidney may be tender, swollen, and less freely movable.

Intermittent hydronephrosis is sometimes associated with movable kidney. Three cases are reported in my Lectures on Abdominal Tumors. In two the condition has been completely relieved by a well-adapted pad and belt; in the third, attacks recur at long intervals.

The *diagnosis* is rarely doubtful, as the shape of the organ is usually distinctive and the mobility marked. Tumors of the gall-bladder, ovarian growths, and tumors of the bowels may in rare instances be confounded with it.

Treatment.—The kidney has been extirpated in many instances, but the operation is not without risk, and there have been several fatal cases. Stitching of the kidney—nephrorrhaphy—as recommended by Hahn, is the most suitable procedure, and statistics published by Keen show that relief is afforded in many cases by the procedure. It does not, however, always succeed.

In many instances the greatest relief is experienced from a bandage and pad. It should be applied in the morning, with the patient in the recumbent posture, and she should be taught how to push up the kidney. An air pad may be used if the organ is sensitive. In other cases a broad bandage well padded in the lower abdominal zone pushes up the intestines and makes them act as a support. In the attacks of severe colic morphia is required. When dependent, as seems sometimes the case, upon an excess of uric acid or the oxalates, the diet must be carefully regulated.

For an exhaustive consideration of all aspects of the subject, see Fischer, in Nos. 1–5 of the Centralblatt f. d. Grenzgebiete der Medicin und Chirurgie, 1898.

III. CIRCULATORY DISTURBANCES.

Normally the secretion of urine is accomplished by the maintenance of a certain blood-pressure within the glomeruli and by the activity of the renal epithelium. Bowman's views on this question have been generally accepted, and the watery elements are held to be filtered from the glomeruli; the amount depending on the rapidity and the pressure of the blood current; the quality, whether normal or abnormal, depending upon the condition of the capillary and glomerular epithelium; while the greater portion of the solid ingredients are excreted by the epithelium of the convoluted tubules. The integrity of the epithelium covering the capillary tufts within Bowman's capsule is essential to the production of a normal urine. If under any circumstances their nutrition fails, as when, for example, the rapidity of the blood current is lowered, so that they are deprived of the necessary amount of oxygen, the material which filters through is no longer normal (i. e., water), but contains serum albumin. Cohnheim has shown that the renal epithelium is extremely sensitive to circulatory changes, and that compression of the renal artery for only a few minutes causes serious disturbance.

The circulation of the kidney is remarkably influenced by reflex stimuli coming from the skin. Exposure to cold causes heightened blood-pressure within the kidneys and increased secretion of urine. Bradford has shown that after excision of portions of the kidney, to as much as one third of the total weight, there is a remarkable increase in the flow of urine.

Congestion of the Kidneys.—(1) *Active Congestion; Hyperæmia.*—Acute congestion of the kidney is met with in the early stage of nephritis, whether due to cold or to the action of poisons and severe irritants. Turpentine, cubebs, cantharides, and copaiba are all stated to cause extreme hyperæmia of the organ. The most typical congestion of the kidney which we see post mortem is that in the early stage of acute Bright's disease, when

the organ may be large, soft, of a dark color, and on section blood drips from it freely.

It has been held that in all the acute fevers the kidneys are congested, and that this explained the scanty, high-colored, and often albuminous urine. On the other hand, by Roy's oncometer, Walter Mendelson has shown that the kidney in acute fever is in a state of extreme anæmia, small, pale, and bloodless; and that this anæmia, increasing with the pyrexia and interfering with the nutrition of the glomerular epithelium, accounts for the scanty, dark-colored urine of fever and for the presence of albumin. In the prolonged fevers, however, it is probable that relaxation of the arteries again takes place. Certainly it is rare to find post mortem such a condition of the kidney as is described by Mendelson. On the contrary, the kidney of fever is commonly swollen, the blood-vessels are congested, and the cortex frequently shows traces of cloudy swelling. However, the circulatory disturbances in acute fevers are probably less important than the irritative effects of either the specific agents of the disease or the products produced in their growth or in the altered metabolism of the tissues. The urine is diminished in amount, and may contain albumin and tube-casts.

(2) *Passive Congestion; Mechanical Hyperæmia.*—This is found in cases of chronic disease of the heart or lung, with impeded circulation, and as a result of pressure upon the renal veins by tumors, the pregnant uterus, or ascitic fluid. In the cardiac kidney, as it is called, the cyanotic induration associated with chronic heart-disease, the organs are enlarged and firm, the capsule strips off, as a rule, readily, the cortex is of a deep red color, and the pyramids of a purple red. The section is coarse-looking, the substance is very firm, and resists cutting and tearing. The interstitial tissue is increased, and there is a small-celled infiltration between the tubules. Here and there the Malpighian tufts have become sclerosed. The blood-vessels are usually thickened, and there may be more or less granular, fatty, or hyaline changes in the epithelium of the tubules. The condition is indeed a diffuse nephritis. The urine is usually reduced, is of high specific gravity, and contains more or less albumin. Hyaline tube-casts and blood-corpuscles are not uncommon. In uncomplicated cases of the cyanotic induration uræmia is rare. On the other hand, in the cardiac cases with extensive arterio-sclerosis, the kidneys are more involved and the renal function is likely to be disturbed.

IV. ANOMALIES OF THE URINARY SECRETION.

1. Anuria.

Total suppression of urine occurs under the following conditions:

(1) As an event in the intense congestion of acute nephritis. For a time no urine may be formed; more often the amount is greatly reduced.

(2) More commonly complete anuria is seen in subjects of renal stone, fragments of which block both ureters. Sir William Roberts calls the con-

dition "latent uræmia." There may be very little discomfort, and the symptoms are very unlike those of ordinary uræmia. Convulsions occurred in only 5 of 41 cases (Herter); headache in only 6; vomiting in only 12. Consciousness is retained; the pupils are usually contracted; the temperature may be low; there are twitchings and perhaps occasional vomiting. Of 41 cases in the literature, 35 occurred in males. Of 36 cases in which there was absolute anuria, in 11 the condition lasted more than four days, in 18 cases from seven to fourteen days, and in 7 cases longer than fourteen days (Herter).

(3) Cases occur occasionally in which the suppression is prerenal. The following are among the more important conditions with which this form of anuria may be associated (Hensley): Fevers and inflammations; acute poisoning by phosphorus, lead, and turpentine; in the collapse after severe injuries or after operations, or, indeed, after the passing of a catheter; in the collapse stage of cholera and yellow fever; and, lastly, there is an hysterical anuria, of which Charcot reports a case in which the suppression lasted for eleven days. Bailey reports the case of a young girl, aged eleven, inmate of an orphan asylum, who passed no urine from October 10th to December 12th (when 8 ounces were withdrawn), and again from this date to March 1st! The question of hysterical deception was considered in the case.

A patient may live for from ten days to two weeks with complete suppression. In Polk's case, in which the only kidney was removed, the patient lived eleven days. It is remarkable that in many instances there are no toxic features. Adams reports a case of recovery after nineteen days of suppression.

In the obstructive cases surgical interference should be resorted to. In the non-obstructive cases, particularly when due to extreme congestion of the kidney, cupping over the loins, hot applications, free purging, and sweating with pilocarpine and hot air are indicated. When the secretion is once started diuretin often acts well. Large hot irrigations, with normal salt solution, with Kemp's double-current rectal tubes, should be tried, as they are stated to stimulate the activity of the kidneys in a remarkable way.

2. Hæmaturia.

The following division may be made of the causes of hæmaturia:

(1) *General Diseases.*—The malignant forms of the acute specific fevers. Occasionally in leukæmia hæmaturia occurs.

(2) *Renal Causes.*—Acute congestion and inflammation, as in Bright's disease, or the effect of toxic agents, such as turpentine, carbolic acid, and cantharides. When the carbolic spray was in use many surgeons suffered from hæmaturia in consequence of this poison. Renal infarction, as in ulcerative endocarditis. New growths, in which the bleeding is usually profuse. In tuberculosis at the onset, when the papillæ are involved, there may be bleeding. Stone in the kidney is a frequent cause. Parasites: The *Filaria sanguinis hominis* and the *Bilharzia* cause a form of hæmaturia met with in the tropics. The echinococcus is rarely associated with hæmorrhage.

(3) *Affections of the Urinary Passages.*—Stone in the ureter, tumor or ulceration of the bladder, the presence of a calculus, parasites, and, very rarely, ruptured veins in the bladder. Bleeding from the urethra occasionally occurs in gonorrhœa and as a result of the lodgment of a calculus.

(4) *Traumatism.*—Injuries may produce bleeding from any part of the urinary passages. By a fall or blow on the back the kidney may be ruptured, and this may be followed by very free bleeding; less commonly the blood comes from injury of the bladder or of the prostate. Blood from the urethra is frequently due to injury by the passage of a catheter, or sometimes to falls or blows.

And, lastly, there is a very interesting group, carefully studied of late years, particularly by Klemperer and M. L. Harris, in which no known lesions have been found. It is probably in this group of cases that Gull's "renal epistaxis" occurs. Harris has recently collected 18 of these cases from the literature. The first-named author thinks it is a form of angioneurotic hæmaturia. An interesting point is that in the 18 cases collected by Harris nephrotomy was done; of these, 9 cases were completely relieved.

Of special interest is the malarial hæmaturia which prevails in certain districts and has already been considered in the section on paludism.

The *diagnosis* of hæmaturia is usually easy. The color of the urine varies from a light smoky to a bright red, or it may have a dark porter color. Examined with the microscope, the blood-corpuscles are readily recognized, either plainly visible and retaining their color, in which case they are usually crenated, or simply as shadows. In ammoniacal urine or urines of low specific gravity the hæmoglobin is rapidly dissolved from the corpuscles, but in normal urine they remain for many hours unchanged.

For other tests the student is referred to the works on Clinical Diagnosis, by Simon and by von Jaksch.

It is important to distinguish between blood coming from the bladder and from the kidneys, though this is not always easy. From the bladder the blood may be found only with the last portions of urine, or only at the termination of micturition. In hæmorrhage from the kidneys the blood and urine are intimately mixed. Clots are more commonly found in the blood from the kidneys, and may form moulds of the pelvis or of the ureter. When the seat of the bleeding is in the bladder, on washing out this organ, the water is more or less blood-tinged; but if the source of the bleeding is higher, the water comes away clear. In many instances it is difficult to settle the question by the examination of the urine alone, and the symptoms and the physical signs must also be taken into account. Cystoscopic examination of the bladder and catheterization of the ureters may aid in the diagnosis in obscure cases.

3. Hæmoglobinuria.

This condition is characterized by the presence of blood-pigment in the urine. The blood-cells are either absent or in insignificant numbers.

In cases of enlarged prostate there may be occasional attacks of haematuria for three or four years preceding the onset of bladder symptoms. (D. Wilson)

The coloring matter is not hæmatin, as indicated by the old name, *hæmatinuria*, nor in reality always hæmoglobin, but it is most frequently methæmoglobin. The urine has a red or brownish-red, sometimes quite black color, and usually deposits a very heavy brownish sediment. When the hæmoglobin occurs only in small quantities, it may give a lake or smoky color to the urine. Microscopical examination shows the presence of granular pigment, sometimes fragments of blood-disks, epithelium, and very often darkly pigmented urates. The urine is also albuminous. The number of red blood-corpuscles bears no proportion whatever to the intensity of the color of the urine. Examined spectroscopically, there are either the two absorption bands of oxyhæmoglobin, which is rare, or, more commonly, there are the three absorption bands of methæmoglobin, of which the one in the red near *C* is characteristic. Two clinical groups may be distinguished.

(1) **Toxic Hæmoglobinuria.**—This is caused by poisons which produce rapid dissolution of the blood-corpuscles, such as chlorate of potash in large doses, pyrogallic acid, carbolic acid, arseniuretted hydrogen, carbon monoxide, naphthol, and muscarine; also the poisons of scarlet fever, yellow fever, typhoid fever, malaria, and syphilis. According to Bastianelli, hæmoglobinuria due to the administration of quinine never occurs excepting in patients who are suffering or who have recently suffered from malarial fever. It has also followed severe burns. Exposure to excessive cold and violent muscular exertion are stated to produce hæmoglobinuria. A most remarkable toxic form occurs in horses, coming on with great suddenness and associated with paresis of the hind legs. Death may occur in a few hours or a few days. The animals are attacked only after being stalled for some days and then taken out and driven, particularly in cold weather. The form of hæmoglobinuria from cold and exertion is extremely rare. No instance of it, even in association with frost-bites, came under my observation in Canada. Blood transfused from one mammal into another causes dissolution of the corpuscles with the production of hæmoglobinuria; and, lastly, there is the *epidemic hæmoglobinuria* of the new-born, associated with jaundice, cyanosis, and nervous symptoms.

(2) **Paroxysmal Hæmoglobinuria.**—This rare disease is characterized by the occasional passage of bloody urine, in which the coloring matter only is present. It is more frequent in males than in females, and occurs chiefly in adults. It seems specially associated with cold and exertion, and has often been brought on, in a susceptible person, by the use of a cold foot-bath. Paroxysmal hæmoglobinuria has been found, too, in persons subject to the various forms of Raynaud's disease. Many regard the relation between these two affections as extremely close; some hold that they are manifestations of one and the same disorder. Druitt, the author of the well-known Surgical Vade-mecum, has given a graphic description of his sufferings, which lasted for many years, and were accompanied with local asphyxia and local syncope. The connection, however, is not very common. In only one of the cases of Raynaud's disease which I have seen was paroxysmal hæmoglobinuria present, and in it epileptic attacks occurred at the same time. The relation of the disease to malaria is not so close as has been

thought by many writers. Bastianelli asserts that it is practically proved that malarial hæmoglobinuria occurs only in infections with the æstivo-autumnal parasite. It rarely, if ever, occurs in the first attack, usually appearing with the first relapse or after repeated relapses. No doubt it has been frequently confounded with a malarial hæmaturia.

The attacks may come on suddenly after exposure to cold or as a result of mental or bodily exhaustion. They may be preceded by chills and pyrexia. In other instances the temperature is subnormal. There may be vomiting and diarrhœa. Pain in the lumbar region is not uncommon. The hæmoglobinuria rarely persists for more than a day or two—sometimes, indeed, not for a day. There are instances in which, even in the course of a single day, there have been two or three paroxysms, and in the intervals clear urine has been passed. Jaundice has been present in a number of cases. According to Ralfe, paroxysmal hæmoglobinuria may alternate with general symptoms of the same character, but associated only with the passage of albumin and an increased quantity of urea in the urine. In such cases he supposes that the toxic agent, whatever its nature, has destroyed only a limited number of the corpuscles, the coloring matter of which is readily dealt with by the spleen and liver, while the globulin is excreted in the urine. The cases are rarely if ever fatal.

The essential pathology of the disease is unknown, and it is difficult to form a theory which will meet all the facts—particularly the relation with Raynaud's disease, which is rightly regarded as a vaso-motor disorder. Increased hæmolysis and solution of the hæmoglobin in the blood-serum (hæmoglobinæmia) precedes, in each instance, the appearance of the coloring matter in the urine. A full discussion of the subject is to be found in F. Chvostek's monograph.

Treatment.—In all forms of hæmaturia rest is essential. In that produced by renal calculi the recumbent posture may suffice to check the bleeding. Full doses of acetate of lead and opium should be tried, then ergot, gallic and tannic acid, and the dilute sulphuric acid. The oil of turpentine, which is sometimes recommended, is a risky remedy in hæmaturia. Extr. hamamelis virgin. and extr. hydrastis canad. are also recommended. Cold may be applied to the loins or dry cups in the lumbar region.

The treatment of hæmoglobinuria is unsatisfactory. Amyl nitrite will sometimes cut short or prevent an attack (Chvostek). During the paroxysm the patient should be kept warm and given hot drinks. Quinine is recommended in large doses, on the supposition—as yet unwarranted—that the disease is specially connected with malaria. If there is a syphilitic history, iodide of potassium in full doses may be tried. In a warm climate the attacks are much less frequent.

4. Albuminuria.

The presence of albumin in the urine, formerly regarded as indicative of Bright's disease, is now recognized as occurring under many circumstances without the existence of serious organic change in the kidney. Two

Lowde (Arch. Gen. de Med. - Sept. 1899) describes a female form of the disease - attacking a number at the time of adolescence.

groups of cases may be recognized—those in which the kidneys show no coarse lesions, and those in which there are evident anatomical changes.

Albuminuria without Coarse Renal Lesions.—(*a*) *Functional, so-called Physiological Albuminuria.*—In a normal condition of the kidney only the water and the salts are allowed to pass from the blood. When albuminous substances transude there is probably disturbance in the nutrition of the epithelium of the capillaries of the tuft, or of the cells surrounding the glomerulus. This statement is still, however, in dispute, and Senator, Grainger Stewart, and others hold that there is a physiological albuminuria which may follow muscular work, the ingestion of food rich in albumin, violent emotions, cold bathing, and dyspepsia. The differences of opinion on this point are striking, and observers of equal thoroughness and reliability have arrived at directly opposite conclusions. The presence of albumin in the urine, in any form and under any circumstance, may be regarded as indicative of change in the renal or glomerular epithelium, a change, however, which may be transient, slight, and unimportant, depending upon variations in the circulation or upon the irritating effects of substances taken with the food or temporarily present, as in febrile states.

Albuminuria of adolescence and cyclic albuminuria, in which the albumin is present only at certain times during the day, are interesting forms. A majority of the cases occur in young persons—boys more commonly than girls—and the condition is often discovered accidentally. The urine, as a rule, contains only a very small quantity of albumin, but in some instances large quantities are present. The most striking feature is the variability. It may be absent in the morning and only present after exertion, or it may be greatly increased after taking food, particularly proteids. The quantity of urine may be but little, if at all, increased, the specific gravity is usually normal, and the color may be high. Occasionally hyaline casts may be found, and in some instances there has been transient glycosuria. As a rule, the pulse is not of high tension and the second aortic sound is not accentuated.

Various forms of this affection have been recognized by writers, such as neurotic, dietetic, cyclic, intermittent, and paroxysmal—names which indicate the characters of the different varieties. A large proportion of the cases get well after the condition has persisted for a variable period. This in itself is an evidence that the changes, whatever their nature, were transient and slight. In these instances the albumin exists in small quantity, tube-casts are rarely present, and the arterial tension is not increased. In a second group the albumin is more persistent, the amount is larger, though it may vary from day to day, and the pulse tension is increased. In such instances the persistent albuminuria probably indicates actual organic change in the kidney.

(*b*) *Febrile Albuminuria.*—Pyrexia, by whatever cause produced, may cause slight albuminuria. The presence of the albumin is due to slight changes in the glomeruli induced by the fever, such as cloudy swelling, which cannot be regarded as an organic lesion. It is extremely common, occurring in pneumonia, diphtheria, typhoid fever, malaria, and even in the fever of acute tonsillitis. The amount of albumin is slight, and it

usually disappears from the urine with the cessation of the fever. Hyaline and even epithelial casts accompany the condition.

(*c*) *Hæmic Changes.*—Purpura, scurvy, chronic poisoning by lead or mercury, syphilis, leukæmia, and profound anæmia may be associated with slight albuminuria. Abnormal ingredients in the blood, such as bile-pigment and sugar, may cause the passage of small amounts of albumin.

The transient albuminuria of pregnancy may belong to this hæmic group, although in a majority of such cases there are changes in the renal tissue. Albumin may be found sometimes after the inhalation of ether or chloroform.

(*d*) Albuminuria occurs in certain affections of the *nervous system.* This so-called neurotic albuminuria is seen after an epileptic seizure and in apoplexy, tetanus, exophthalmic goitre, and injuries of the head.

Albuminuria with Definite Lesions of the Urinary Organs.—(*a*) Congestion of the kidney, either active, such as follows exposure to cold and is associated with the early stages of nephritis, or passive, due to obstructed outflow in disease of the heart or lungs, or to pressure on the renal veins by the pregnant uterus or tumors.

(*b*) Organic disease of the kidneys—acute and chronic Bright's disease, amyloid and fatty degeneration, suppurative nephritis, and tumors.

(*c*) Affections of the pelvis, ureters, and bladder, when associated with the formation of pus.

Tests for Albumin.—Both morning and evening urine should be examined, and in doubtful cases at least three specimens. If turbid, the urine should be filtered, though turbidity from the urates is of no moment, since it disappears at once on the application of heat.

Heat and Nitric-acid Test.—The urine is boiled in a test-tube over a spirit-lamp, and a drop of nitric acid is then added. If a cloudiness occurs on boiling, it may be due to phosphates, which are dissolved on the addition of an acid. Persistence of the cloudiness indicates albumin.

Heller's Test.—A small quantity of fuming nitric acid is poured into the test-tube, and with a pipette the urine is allowed to flow gently down the side upon the acid. At the line of junction of the two fluids, if albumin is present, a white ring is formed. This contact method is trustworthy, and, for the routine clinical work, is probably the most satisfactory. A diffused haze, due to mucin (nucleo-albumin), is sometimes seen just above the white ring of albumin; and in very concentrated urines, or after the taking of balsamic remedies, a slight cloudiness may be due to urates or uric acid, which clears on heating or warming. A colored ring at the junction of the acid and the urine is due to the oxidation of the coloring matters in the urine.

Ferrocyanide-of-potassium and Acetic-acid Test.—Fill an ordinary test-tube half full of urine, and add 5 or 6 cc. of potassium-ferrocyanide solution (1 in 20). Thoroughly mix the urine and reagent and add 10 to 15 drops of acetic acid. If albumin be present, a cloudiness varying in degree according to the amount of albumin will be produced. This is a very reliable test, as it precipitates all forms of albumin, acid and alkaline, but

does not precipitate mucin, peptones, phosphates, urates, vegetable alkaloids, or the pine acids.

Sir William Roberts strongly recommends the *magnesium-nitric test.* One volume of strong nitric acid is mixed with five volumes of the saturated solution of sulphate of magnesium. This is used in the same way as the nitric acid in Heller's test.

Picric acid, introduced by George Johnson, is a delicate and useful test for albumin. A saturated solution is used and employed as in the contact method. It has been urged against this test that it throws down the mucin, peptones, and certain vegetable alkaloids, but these are dissolved by heat.

For minute traces of albumin the trichloracetic acid may be used, or Millard's fluid, which is extremely delicate and consists of glacial carbolic acid (95 per cent), 2 drachms; pure acetic acid, 7 drachms; liquor potassæ, 2 ounces 6 drachms.

A quantitative estimate of the albumin can be made by means of Esbach's tube, but the rough method of heating and boiling a certain quantity of acidulated urine in a test-tube and allowing it to stand, is often employed. The depth of deposit can then be compared with the whole amount of urine, and the proportion is expressed as a mere trace, almost solid—one fourth, one half, and so on. This, of course, does not give an accurate indication of the proportion of albumin in the total quantity of urine. For the more elaborate methods the reader is referred to the works on urinalysis.

The above tests refer entirely to serum albumin. Other albuminous substances occur, such as albumose, serum globulin, peptones, and hemialbumose or propepton. They are not of much clinical importance.

Albumosuria.—Traces of *peptones* (albumoses) are found in the urine in many febrile diseases and in chronic suppuration. Albumosuria has but little clinical significance except in one connection. In 1848 Bence-Jones described a case of osteo-malacia in which he found a modified form of albumin in the urine. Of late years renewed interest has been taken in the subject by the discovery of the association of albumose with multiple myelomata of the bones. As Kahler called special attention to it, the Italians have given the condition his name. Fitz reported an instance at the last meeting (1898) of the Association of American Physicians, the only one recognized, so far as I know, in this country. In Bradshaw's case the patient passed at intervals for a year a turbid, milky urine, which deposited a copious white sediment. On adding nitric acid to a urine containing albumose a white precipitate is formed, which is dissolved when the specimen is boiled, but reappears on cooling.

Globulin rarely occurs in the urine alone, but generally in association with serum-albumin. The latter is usually present in greater quantity, but in severe organic renal disease and in diabetes Maguire has found that the proportion of globulin to albumin is often 2.5 to 1. Senator states that more globulin is present in lardaceous kidney than in other forms of nephritis. The clinical significance of globulin is the same as that of serum-albumin.

Prognosis.—This depends, of course, entirely upon the cause. Febrile albuminuria is transient, and in a majority of the cases depending upon hæmic causes the condition disappears and leaves the kidneys intact. An occasional trace of albumin in a man over forty, with or without a few hyaline casts, and with increased tension and thick vessel walls, usually indicates changes in the kidneys. The persistence of a slight amount of albumin in young men without increased arterial tension is less serious, as even after continuing for years it may disappear. I have already spoken of the outlook in the so-called cyclic albuminuria.

Practically in all cases the presence of albumin indicates a change of some sort in the glomeruli, the nature, extent, and gravity of which it is difficult to estimate; so that other considerations, such as the presence of tube-casts, the existence of increased tension, the general condition of the patient, and the influence of digestion upon the albumin, must be carefully considered.

The physician is daily consulted as to the relation of albuminuria and life assurance. As his function is to protect the interests of the company, he should reject all cases in which albumin occurs in the urine. It is even doubtful if an exception should be made in young persons with transient albuminuria. Naturally, companies lay great stress upon the presence or absence of albumin, but in the most serious and fatal malady with which they have to deal—chronic interstitial nephritis—the albumin is often absent or transient, even when the disease is well developed. After the fortieth year, from a standpoint of life insurance, the state of the arteries is far more important than the condition of the urine.

With reference to the significance of albuminuria in adults, I quite agree with the following conclusions of F. C. Shattuck:

(1) Renal albuminuria, as proved by the presence of both albumin and casts, is much more common in adults, quite apart from Bright's disease or any obvious source of renal irritation, than is generally supposed.

(2) The frequency increases steadily and progressively with advancing age.

(3) This increase with age suggests the explanation that the albuminuria is often an indication of senile degeneration.

(4) Though it cannot be regarded as yet as absolutely proved, it is highly probable that faint traces of albumin and hyaline and finely granular casts of small diameter are often, especially in those past fifty years of age, of little or no practical importance.

5. Pyuria (*Pus in the Urine*).

Causes.—(1) *Pyelitis and Pyelonephritis.*—In large abscesses of the kidney, pyonephrosis, the pus may be intermittent, and for days or even weeks the urine is free. In calculous and tuberculous pyelitis the pyuria is usually continuous, though varying in intensity. In these cases, as a rule, the pus is mixed with the urine, which is acid in reaction. In the early stages of pyelitis the transitional epithelium may be abundant, but is not in any way distinctive. In the pyelitis and pyelonephritis following cystitis the

urine is usually alkaline, and contains more mucus; micturition is usually more frequent, and the history points to a previous bladder affection.

(2) *Cystitis.*—The urine is alkaline, often fetid, the pus ropy, and the amount of urine greatly increased. The ropy, thick mucus usually comes with the last portions of the urine. Triple phosphate crystals may be present in the freshly passed urine.

(3) *Urethritis*, particularly gonorrhœa. The pus appears first, is in small quantities, and there are signs of local inflammation.

(4) In *leucorrhœa* the quantity of pus is usually small, and large flakes of vaginal epithelium are numerous. In doubtful cases, when leucorrhœa is present, the urine should be withdraw through a catheter.

(5) *Rupture of Abscesses into the Urinary Passages.*—In such cases as pelvic or perityphlitic abscess there have been previous symptoms of pus formation. A large amount is passed within a short time, then the discharge stops abruptly or rapidly diminishes within a few days.

Pus gives to the urine a white or yellowish-white appearance. On settling there is a heavy grayish sediment, and the supernatant fluid is usually turbid. The sediment is often tenacious and ropy. The reaction is generally alkaline, and the odor may be ammoniacal even when passed. Examination with the microscope reveals the presence of a large number of pus-corpuscles, which are usually, when the pus comes from the bladder, well formed; the protoplasm is granular, and often shows many translucent processes.

The only sediment likely to be confounded with pus is that of the phosphates; but it is whiter and less dense, and is distinguished immediately by microscopical examination.

With the pus there is always more or less epithelium from the bladder and pelves of the kidneys, but since in these situations the forms of cells are practically identical, they afford no information as to the locality from which the pus has come.

The treatment of pus in the urine is considered under the conditions in which it occurs.

6. Chyluria—Non-parasitic.

This is a rare affection, occurring in temperate regions and unassociated with the *Filaria Bancrofti*. The urine is of an opaque white color; it resembles milk closely, is occasionally mixed with blood (hæmatochyluria), and sometimes coagulates into a firm, jelly-like mass. In other instances there is at the bottom of the vessel a loose clot which may be distinctly blood-tinged. Under the microscope the turbidity seems to be caused by numerous minute granules—more rarely oil droplets similar to those of milk. In Montreal I made the dissection of a case of thirteen years' duration and could find no trace of parasites.

7. Lithuria (*Lithæmia; Lithic-acid Diathesis*).

The general relations of uric acid have already been considered in speaking of gout.

Occurrence in the Urine.—The uric acid occurs in combination chiefly with ammonium and sodium, forming the acid urates. In smaller quantities are the potassium, calcium, and lithium salts. The uric acid may be separated from its bases and crystallizes in rhombs or prisms, which are usually of a deep red color, owing to the staining of the urinary pigments. The sediment formed is granular and the groups of crystals look like grains of Cayenne pepper. It is very important not to mistake a deposit of uric acid for an excess. The deposition of numerous grains in the urine within a few hours after passing is more likely to be due to conditions which diminish the solvent power than to increase in the quantity. Of the conditions which cause precipitation of the uric acid Roberts gives the following: "(1) High acidity; (2) poverty in mineral salts; (3) low pigmentation; and (4) high percentage of uric acid." The grade of acidity is probably the most important element.

In health the weight of uric acid excreted bears a fairly constant ratio to the weight of urea eliminated. According to von Noorden, the average ratio is 1 to 50, while the average ratio of the nitrogen of uric acid to the total nitrogen eliminated in the urine is 1 to 70. In several of the cases of gout in my wards Futcher found that in the intervals between the acute arthritic attacks the uric acid was reduced to a much greater extent than the urea, so that the ratio of the former to the latter often varied between 1 to 300 up to (in one case) 1 to 1,500, a return to about the normal proportions occurring during the acute attacks.

More common is the precipitation of amorphous urates, forming the so-called brick-dust or lateritious deposit, which has a pinkish color, due to the presence of urinary pigment. It is composed chiefly of the acid sodium urates. It occurs particularly in very acid urine of a high specific gravity. As the urates are more soluble in warm solutions, they frequently deposit as the urine cools. Here, too, the deposition does not necessarily, indeed usually does not, mean an excessive excretion, but the existence of conditions favoring the deposit.

Lithæmia.—In addition to what has already been said under *gout*, we may consider here the hypothetical condition known as lithæmia, or the uric-acid diathesis. Murchison introduced the term to designate certain symptoms due, as he supposed, to functional disturbance of the liver. Not only have his views been widely adopted, but, as is so often the case when we give the rein to theoretical conceptions of disease, the so-called manifestations of this state have so multiplied that some authors attribute to this cause a considerable proportion of the ailments affecting the various systems of the body. Thus one writer enumerates not fewer than thirty-nine separate morbid conditions associated with lithæmia! From our lack of knowledge of the mode of formation and elimination of uric acid it is very evident that the physiology of the subject must be widely extended before we are in a position to draw safe conclusions. Thus it is by no means sure that, as Murchison supposed, the essential defect is in a functional disorder of the liver, disturbing the metabolism of the albuminous ingredients, nor is it at all certain that the only offending substance is uric acid. In the present imperfect state of knowledge it is impossible with

The discovery of oxalate of lime crystals in the urine by Donné in 1838 led to the description of the so-called Oxalic acid diathesis. It is claimed that all the oxalic acid found in the urine is taken into the body with the food. (Dunlop) In health normally only a trace is formed in the body. The amount fluctuates with the quantity of food taken & is ~~from 01 to 02 a few milligrammes~~ usually below 16 milligrammes daily (Baldwin) It seems to be formed in the body when there is an absence of free hydrochloric acid in the gastric juice & in connection with excessive fermentation in the intestines. Clinically it has two points of interest. (a)

6

any clearness to define the pathology of the so-called uric-acid diathesis. We may say that certain symptoms arise in connection with defective food or tissue metabolism, more particularly of the nitrogenous elements. Deficient oxidation is probably the most essential factor in the process, with the result of the formation of less readily soluble and less readily eliminated products of retrograde metamorphosis. This faulty metabolism if long continued may lead to gout, with uratic deposits in the joints, acute inflammations, and arterial and renal disease. In a large group of cases the disturbed metabolism produces high tension in the arteries (probably as a direct sequence of interference with the capillary circulation) and ultimately degenerations in various tissues, particularly the scleroses.

Overeating and overdrinking, when combined with deficient muscular exercise, lie at the basis of this nutritional disturbance. The symptoms which are believed to characterize the uric-acid diathesis have already been briefly treated of under the section on irregular gout, and the question of diet and exercise has also been there considered.

8. Oxaluria.

Oxalic acid occurs in the urine, in combination with lime, forming an oxalate which is held in solution by the acid phosphate of soda. About .01 to .02 gramme is excreted in the day. It never forms a heavy deposit, but the crystals—usually octahedral, rarely dumb-bell-shaped—collect in the mucus-cloud and on the sides of the vessel. The amount varies extremely with the diet, and it is increased largely when such fruits and vegetables as tomatoes and rhubarb are taken. It is also a product of incomplete oxidation of the organic substances in the body, and in conditions of increased metabolism the amount in the urine becomes larger. It is stated also to result from the acid fermentation of the mucus in the urinary passages, and the crystals are usually abundant in spermatorrhœa.

When in excess and present for any considerable time, the condition is known as oxaluria, the chief interest of which is in the fact that the crystals may be deposited before the urine is voided, and form a calculus. It is held by many that there is a special diathesis associated with this state and manifested clinically by dyspepsia, particularly the nervous form, irritability, depression of spirits, lassitude, and sometimes marked hypochondriasis. There may be in addition neuralgic pains and the general symptoms of neurasthenia. The local and general symptoms are probably dependent upon some disturbance of metabolism of which the oxaluria is one of the manifestations. It is a feature also in many gouty persons, and in the condition called lithæmia.

9. Cystinuria.

Stadthogen claims that normal urine does not contain cystin, though Baumann and Goldmann succeeded in separating it in very small quantities from healthy urine as a benzoyl compound. It is associated with elimination of diamines both in the fæces and urine. It is very rarely met

with, and its chief interest is owing to the fact that it may form a calculus. Its presence in the urine has been determined in many members of the same family, and the condition appears sometimes to be hereditary. As it contains sulphur, it is thought to be formed from the taurin of the bile.

10. Phosphaturia.

The phosphoric acid is excreted from the body in combination with potassium, sodium, calcium, and magnesium, forming two classes, the alkaline phosphates of sodium and potassium and the earthy phosphates of lime and magnesia. The amount of phosphoric acid (P_2O_5) excreted in the twenty-four hours varies, according to Hammarsten, between 1 and 5 grammes, with an average of 2.5 grammes. It is derived mainly from the phosphoric acid taken in the food, but also in part as a decomposition product from nuclein, protagon, and lecithin. Of the alkaline phosphates, those in combination with sodium are the most abundant. The alkaline phosphates of the urine are more abundant than the earthy phosphates.

Of the *earthy phosphates*, those of lime are abundant, of magnesium scanty. In urine which has undergone the ammoniacal fermentation, either inside or outside the body, there is in addition the ammonio-magnesium or triple phosphate, which occurs in triangular prisms or in feathery or stellate crystals; hence the term given to this form of stellar phosphates. The earthy phosphates occur as a sediment in the urine when the alkalinity is due to a fixed alkali, or under certain circumstances the deposit may take place within the bladder, and then the phosphates are passed at the end of micturition as a whitish fluid, which is popularly confounded with spermatorrhœa. The calcium phosphate may be precipitated by heat and produce a cloudiness which may be mistaken for albumin, but is at once dissolved upon making the urine acid. This condition is very frequent in persons suffering from dyspepsia or from debility of any kind. The phosphates may be in great excess, rising in the twenty-four hours to from 7 to 9 grammes (Tessier), whereas the normal amount is not more than 2.5 grammes. And, lastly, the phosphates may be deposited in urine which has undergone decomposition, in which the carbonate of ammonia from the urea combines with the magnesium phosphates, forming the triple salt. This is seen in cystitis, and is due to the introduction of a bacterial ferment.

The clinical significance of an excess of phosphates, to which the term phosphaturia is applied, has been much discussed. It must be remembered that a deposit does not necessarily mean an excess, to determine which a careful analysis of the twenty-four hours' secretion should be made. It has long been thought that there is a relation between the activity of the nerve-tissues and the output of phosphoric acid; but the question cannot yet be considered settled. The amount is increased in wasting diseases, such as phthisis, acute yellow atrophy of the liver, leukæmia, and severe anæmia, whereas it is diminished in acute diseases and during pregnancy.

In a condition termed by Tessier, Ralfe, and others, phosphatic diabetes there are polyuria, thirst, emaciation, and a great increase in the

excretion of phosphates, which may be as much as from 7 to 9 grammes in the day. The urine is usually acid and free from sugar; the patients are nervous; in some instances sugar has been present in the urine, and in others it subsequently makes its appearance.

11. Indicanuria.

The substance in the urine which has received this name is the indoxyl-sulphate of potassium, in which form it appears in the urine and is colorless. When concentrated acids or strong oxidizing agents are added to the urine, this substance is decomposed and the indigo set free. It is present only in small quantities in healthy urine. It is derived from the indol, a product formed in the intestine by the decomposition of the albumin under the influence of bacteria. When absorbed, this is oxidized in the tissues to indoxyl, which combines with the potassium sulphate, forming the above-named substance.

The quantity of indican is diminished on a milk (and a Kefir) diet. It is increased in all wasting diseases, as carcinoma, and whenever any large quantities of albuminous substances are undergoing rapid decomposition, as in the severer forms of peritonitis and empyema. It is not usually increased in constipation, but is met with in ileus, particularly in obstruction of the small intestine. Indican has occasionally been found in calculi. Though, as a rule, the urine is colorless when passed, there are instances in which the decomposition has taken place within the body, and a blue color has been noticed immediately after the urine was voided. Sometimes, too, in alkaline urine on exposure there is a bluish film on the surface.

To test for indican, place 4 or 5 cc. of nitric or hydrochloric acid in a test-tube; boil, and add an equal quantity of urine. A bluish ring develops at the point of contact. Add 1 or 2 cc. of chloroform and shake the test-tube; on separation the chloroform has a violet or bluish color due to the presence of indican.

12. Melanuria.

In melanotic cancer the urine, either at the time of voiding or after exposure to the air, may present a dark color. This pigment is known as melanin, and it may occur in solution or in the form of small granules. The urine may be voided clear, and subsequently, on exposure to the air or on the addition of oxidizing substances, becomes dark. In these cases it contains a chromogen called melanogen, which turns dark by oxidation. Von Jaksch has found that "in urine containing melanin or its precursor, melanogen, Prussian blue is formed by adding a nitroprusside, aqueous potash, and an acid. This reaction, however, does not seem to depend on the presence of melanin, as it is not given by that substance when separated from the urine, but apparently by some other at present unknown substance, which is present in traces in normal urine and is increased in cases of melanuria, and also in those conditions where excess of indigo occurs in the urine" (Halliburton).

13. Pneumaturia.

Gas may be passed with the urine—

1. After mechanical introduction of air in vesical irrigation or cystoscopic examination in the knee-elbow position.

2. As a result of the introduction of gas-forming organisms in catheterization or other operation. Glycosuria has been present in a majority of the cases. The yeast fungus, the colon bacillus, and the bacillus aërogenes capsulatus have been found.

3. In cases of vesico-enteric fistula.

In gas production within the bladder the symptoms are those of a mild cystitis, with the passage of gas at the end of micturition, sometimes with a loud sound. The diagnosis is readily made by causing the patient to urinate in a bath or by plunging the end of the catheter under water.

14. Other Substances.

Fat in the urine, or *lipuria*, occurs, according to Halliburton, first, without disease of the kidneys, as in excess of fat in the food, after the administration of cod-liver oil, in fat embolism occurring after fractures, in the fatty degeneration in phosphorus poisoning, in prolonged suppuration, as in phthisis and pyæmia, in the lipæmia of diabetes mellitus; secondly, with disease of the kidneys, as in the fatty stage of chronic Bright's disease, in which fat casts are sometimes present, and, according to Ebstein, in pyonephrosis; and, thirdly, in the affection known as chyluria. The urine is usually turbid, but there may be fat drops as well, and fatty crystals have been found.

Lipaciduria is a term applied by von Jaksch to the condition in which there are volatile fatty acids in the urine, such as acetic, butyric, formic, and propionic acid.

Acetonuria.—Von Jaksch distinguishes the following forms of pathological acetonuria: The febrile, the diabetic, the acetonuria with certain forms of cancer, the form associated with inanition, acetonuria in psychoses, and the acetonuria which results from auto-intoxication. It is doubtful, however, whether the symptoms in these are really due to the acetone. It may be the substances from which this is formed, particularly the diacetic acid or the β-oxy-butyric acid. The odor of the acetone may be marked in the breath and evident in the urine. The tests have been given in the section on diabetes.

Diacetic acid is probably never present in the urine in health. With a solution of ferric chloride it gives a Burgundy-red color. A similar reaction is given by acetic, formic, and oxy-butyric acids; it may be present in the urine of patients who are taking antipyrin, thallin, and the salicylates. Hammarsten states that if the reaction be due to the presence of diacetic acid, it will not be obtained in carrying out the test with a second specimen of urine which has been boiled and allowed to cool. The ethereal extract of the acidulated urine gives the reaction if diacetic acid be present, whereas the other substances which may be mistaken for diacetic acid are insoluble in ether.

β-oxy-butyric acid is believed by Stadelmann, Külz, and Minkowski to be the cause of diabetic coma. It is a product of the decomposition of the tissue albumins, and from it diacetic acid is readily formed by oxidation. Its tests have already been given.

Alcaptonuria.—Aromatic compounds occur after the administration of carbolic acid or gallic acid, and the urine on exposure to air becomes dark. In carboluria the substance causing the black color is known as hydrochinon. Many years ago Boedeker met with cases in which the urine became dark, owing to the presence of an aromatic compound which he called alcapton. The urine is clear on passing, and then darkens on exposure to the air, or on the addition of liquor potassæ. Baumann isolated a substance from the urine of a case of alcaptonuria, to which he gave the name of homogentisinic acid. Later observers have isolated this substance in other cases. Kirk believed the reaction in his case was due to uroleucinic acid. In several instances more than one member of a family has shown this urinary change. The substance is apparently without clinical significance except in so far as it is capable of reducing the Fehling solution, and may be mistaken for sugar. Alcapton urine may be distinguished from diabetic urine from the fact that it does not ferment nor reduce alkaline bismuth solutions, and because it is optically inactive (see Alcaptonuria, by T. B. Futcher, New York Med. Jour., 1897, ii).

Choluria and glycosuria have already been considered under jaundice and diabetes.

Hæmatoporphyrin occasionally occurs in the urine. It was first recognized by Hoppe-Seyler. Nencki and Sieler determined its exact formula, and the former demonstrated that the only chemical difference between hæmatin and hæmatoporphyrin is that the latter is simply hæmatin free from iron. It has been found in the urine in pulmonary tuberculosis, pleurisy with effusion, acute rheumatism, lead poisoning, and intestinal hæmorrhages. This pigment has been found very frequently after the administration of sulphonal, and sometimes imparts a very dark color to the urine.

V. URÆMIA.

Definition.—A toxæmia developing in the course of nephritis or in conditions associated with anuria. The nature of the poison or poisons is as yet unknown, whether they are the retained normal products or the products of an abnormal metabolism.

Theories of Uræmia.—The view most widely held is that uræmia is due to the accumulation in the blood of excrementitious material—body poisons—which should be thrown off by the kidneys. "If, however, from any cause, these organs make default, or if there be any prolonged obstruction to the outflow of urine, accumulation of some or of all the poisons takes place, and the characteristic symptoms are manifested, but the accumulation may be very slow and the earlier symptoms, corresponding to the comparatively small dose of poison, may be very slight; yet they are in kind, though not in degree, as indicative of uræmia as are the more alarm-

ing, which appear toward the end, and to which alone the name uræmia is often given" (Carter). Herter and others have shown that the toxicity of the blood-serum in uræmic states is increased. The part played by urea itself, by the salts, and by the nitrogenous extractives has not been determined.

Another view is that uræmia depends on the products of an abnormal metabolism. Brown-Séquard suggested that the kidney has an internal secretion, and it is urged that the symptoms of uræmia are due to its disturbance. Bradford's experiments show that the kidneys do influence profoundly the metabolism of the tissues of the body, particularly of the muscles. If more than one third of the total kidney weight be removed, there is an extraordinary increase in the production of urea and of the nitrogenous bodies of the creatin class. He favors this view, but acknowledges that we are still ignorant of the nature of the poison. From a careful study of the question, Hughes and Carter concluded that the poison was an albuminous product quite different from anything in normal urine. In Bradford's Gulstonian Lectures (1898) will be found a full discussion of the question.

Traube believed that the symptoms of uræmia, particularly the coma and convulsions, were due to localized œdema of the brain.

Symptoms.—Clinically, we may recognize latent, acute, and chronic forms of uræmia. The latent form has been considered under the section on anuria. Acute uræmia may develop in any form of nephritis. It is more common in the post-febrile varieties. Bradford thinks that it is specially associated with a form of contracted white kidney in young subjects. Chronic forms of uræmia are more frequent in the arterio-sclerotic and granular kidney. For convenience the symptoms of uræmia may be described under cerebral, dyspnœic, and gastro-intestinal manifestations.

Among the *cerebral* symptoms of uræmia may be described:

(*a*) *Mania.*—This may come on abruptly in an individual who has shown no previous indications of mental trouble, and who may not be known to have Bright's disease. In a remarkable case of this kind which came under my observation the patient became suddenly maniacal and died in six days. More commonly the delirium is less violent, but the patient is noisy, talkative, restless, and sleepless.

(*b*) *Delusional Insanity* (*Folie Brightique*).—Cases are by no means uncommon, and excellent clinical reports have been issued on the subject from several of the asylums of this country, particularly by Bremer, Christian, and Alice Bennett. Delusions of persecution are common. One of my cases committed suicide by jumping out of a window. The condition is of interest medico-legally because of its bearing on testamentary capacity. Profound melancholia may also supervene.

(*c*) *Convulsions.*—These may come on unexpectedly or be preceded by pain in the head and restlessness. The attacks may be general and identical with those of ordinary epilepsy, though the initial cry may not be present. The fits may recur rapidly, and in the interval the patient is usually unconscious. Sometimes the temperature is elevated, but more frequently it is depressed, and may sink rapidly after the attack. Local or Jacksonian epilepsy may occur in most characteristic form in uræmia.

A remarkable sequence of the convulsions is blindness—*uræmic amaurosis*—which may persist for several days. This, however, may occur apart from the convulsions. It usually passes off in a day or two. There are, as a rule, no ophthalmoscopic changes. Sometimes uræmic deafness supervenes, and is probably also a cerebral manifestation. It may also occur in connection with persistent headache, nausea, and other gastric symptoms.

(*d*) *Coma.*—Unconsciousness invariably accompanies the general convulsions, but a coma may develop gradually without any convulsive seizures. Frequently it is preceded by headache, and the patient gradually becomes dull and apathetic. In these cases there may have been no previous indications of renal disease, and unless the urine is examined the nature of the case may be overlooked. Twitchings of the muscles occur, particularly in the face and hands, but there are many cases of coma in which the muscles are not involved. In some of these cases a condition of torpor persists for weeks or even months. The tongue is usually furred and the breath very foul and heavy.

(*e*) *Local Palsies.*—In the course of chronic Bright's disease hemiplegia or monoplegia may come on spontaneously or follow a convulsion, and post mortem no gross lesions of the brain be found, but only a localized or diffused œdema. These cases, which are not very uncommon, may simulate almost every form of organic paralysis of cerebral origin.

(*f*) Of other cerebral symptoms, headache is important. It is most often occipital and extends to the neck. It may be an early feature and associated with giddiness. Other nervous symptoms of uræmia are intense itching of the skin, numbness and tingling in the fingers, and cramps in the muscles of the calves, particularly at night. An erythema may be present.

Uræmic dyspnœa is classified by Palmer Howard as follows: (1) Continuous dyspnœa; (2) paroxysmal dyspnœa; (3) both types alternating; and (4) Cheyne-Stokes breathing. The attacks of dyspnœa are most commonly nocturnal; the patient may sit up, gasp for breath, and evince as much distress as in true asthma. Occasionally the breathing is noisy and stridulous. The Cheyne-Stokes type may persist for weeks, and is not necessarily associated with coma. I have seen it in a man who travelled over a hundred miles to consult a physician. In another instance a patient, up and about, could only when at meals feed himself in the apnœa period. Though usually of serious omen and occurring with coma and other symptoms, recovery may follow even after persistence for weeks or even months.

The *gastro-intestinal* manifestations of uræmia often set in with abruptness. Uncontrollable vomiting may come on and its cause be quite unrecognizable. A young married woman was admitted to my wards in the Montreal General Hospital with persistent vomiting of four or five days' duration. The urine was slightly albuminous, but she had none of the usual signs of uræmia, and the case was not regarded as one of Bright's disease. The vomiting persisted and caused death. The post mortem showed extensive sclerosis of both kidneys. The attacks may be preceded by nausea and may be associated with diarrhœa. In some instances the diarrhœa may come on without the vomiting; sometimes it is profuse and

associated with an intense catarrhal or even diphtheritic inflammation of the colon.

A special uræmic *stomatitis* has been described (Barie) in which the mucosa of the lips, gums, and tongue is swollen and erythematous. The saliva may be increased, and there is difficulty in swallowing and in mastication. The tongue is usually very foul and the breath heavy and fetid. A cutaneous erythema may be present in uræmia.

Fever is not uncommon in uræmic states, and may occur with the acute nephritis, with the complications, and as a manifestation of the uræmia itself (Stengel).

Very many patients with chronic uræmia succumb to what I have called terminal infections—acute peritonitis, pericarditis, pleurisy, meningitis, or endocarditis.

Diagnosis.—Herter calls attention to the value of the clinical determination of the urea in the blood (for which purpose only a few cubic centimetres are required) as an index of the degree of renal inadequacy. So far as the urine is concerned, the volume and specific gravity indicate the total solids, and the determination of the urea itself in the urine gives no indication of the quantity in the blood. Uræmia may be confounded with:

(*a*) Cerebral lesions, such as hæmorrhage, meningitis, or even tumor. In apoplexy, which is so commonly associated with kidney disease and stiff arteries, the sudden loss of consciousness, particularly if with convulsions, may simulate a uræmic attack; but the mode of onset, the existence of complete hemiplegia, with conjugate deviation of the eyes, suggest hæmorrhage. As already noted, there are cases of uræmic hemiplegia or monoplegia which cannot be separated from those of organic lesion and which post mortem show no trace of coarse disease of the brain. I know of an instance in which a consultation was held upon the propriety of operation in a case of hemiplegia believed to be due to subdural hæmorrhage which post mortem was shown to be uræmic. Indeed, in some of these cases it is quite impossible to distinguish between the two conditions. So, too, cases of meningitis, in a condition of deep coma, with perhaps slight fever, furred tongue, and without localizing symptoms, may readily be confounded with uræmia.

(*b*) With certain infectious diseases. Uræmia may persist for weeks or months and the patient lies in a condition of torpor or even unconsciousness, with a heavily coated, perhaps dry, tongue, muscular twitchings, a rapid feeble pulse, with slight fever. This state not unnaturally suggests the existence of one of the infectious diseases. Cases of the kind are not uncommon, and I have known them to be mistaken for typhoid fever and for miliary tuberculosis.

(*c*) Uræmic coma may be confounded with poisoning by alcohol or opium. In opium poisoning the pupils are contracted; in alcoholism they are more commonly dilated. In uræmia they are not constant; they may be either widely dilated or of medium size. The examination of the eye-ground should be made to determine the presence or absence of albuminuric retinitis. The urine should be drawn off and examined. The odor of the breath sometimes gives an important hint.

The condition of the heart and arteries should also be taken into account. Sudden uræmic coma is more common in the chronic interstitial nephritis. The character of the delirium in alcoholism is sometimes important, and the coma is not so deep as in uræmia or opium poisoning. It may for a time be impossible to determine whether the condition is due to uræmia, profound alcoholism, or hæmorrhage into the pons Varolii.

And lastly, in connection with sudden coma, it is to be remembered that insensibility may occur after prolonged muscular exertion, as after running a ten-mile race. In some instances unconsciousness has come on rapidly with stertorous breathing and dilated pupils. Cases have occurred under conditions in which sun-stroke could be excluded; and Poore, who reports a case in the Lancet (1894), considers that the condition is due to the too rapid accumulation of waste products in the blood, and to hyperpyrexia from suspension of sweating.

The treatment will be considered under Chronic Bright's Disease.

VI. ACUTE BRIGHT'S DISEASE.

Definition.—Acute diffuse nephritis, due to the action of cold or of toxic agents upon the kidneys.

In all instances changes exist in the epithelial, vascular, and intertubular tissues, which vary in intensity in different forms; hence writers have described a tubular, a glomerular, and an acute interstitial nephritis. Delafield recognizes *acute exudative* and *acute productive* forms, the latter characterized by proliferation of the connective-tissue stroma and of the cells of the Malpighian tufts.

Etiology.—The following are the principal causes of acute nephritis:

(1) Cold. Exposure to cold and wet is one of the most common causes. It is particularly prone to follow exposure after a drinking-bout.

(2) The poisons of the specific fevers, particularly scarlet fever, less commonly typhoid fever, measles, diphtheria, small-pox, chicken-pox, malaria, cholera, yellow fever, meningitis, and, very rarely, dysentery. As already mentioned, acute nephritis may be associated with syphilis. In acute tuberculosis nephritis is not uncommon. It may also occur in septicæmia. The frequency of acute nephritis in malaria has been emphasized by Thayer in a recent analysis of the cases at the Johns Hopkins Hospital. Among 1,832 cases there were 26 of nephritis.

(3) Toxic agents, such as turpentine, cantharides, chlorate of potash, and carbolic acid may cause an acute congestion which sometimes terminates in nephritis. Alcohol probably never excites an acute nephritis.

(4) Pregnancy, in which the condition is thought by some to result from compression of the renal veins, although this is not yet finally settled. The condition may in reality be due to toxic products as yet undetermined.

(5) Acute nephritis occurs occasionally in connection with extensive lesions of the skin, as in burns or in chronic skin-diseases.

Morbid Anatomy.—The kidneys may present to the naked eye in mild cases no evident alterations. When seen early in more severe forms

the organs are congested, swollen, dark, and on section may drip blood. In other instances the surface is pale and mottled, the capsule strips off readily, and the cortex is swollen, turbid, and of a grayish-red color, while the pyramids have an intense beefy-red tint. The glomeruli in some instances stand out plainly, being deeply swollen and congested; in other instances they are pale.

The histology may be thus summarized: (*a*) Glomerular changes. In a majority of the cases of nephritis due to toxic agents, which reach the kidney through the blood-vessels, the tufts suffer first, and there is either an acute intracapillary glomerulitis, in which the capillaries become filled with cells and thrombi, or involvement of the epithelium of the tuft and of Bowman's capsule, the cavity of which contains leucocytes and red blood-corpuscles. Hyaline degeneration of the contents and of the walls of the capillaries of the tuft is an extremely common event. These processes are perhaps best marked in scarlatinal nephritis. There may be proliferation about Bowman's capsule. These changes interfere with the circulation in the tufts and seriously influence the nutrition of the tubular structures beyond them.

(*b*) The alterations in the tubular epithelium consist in cloudy swelling, fatty change, and hyaline degeneration. In the convoluted tubules, the accumulation of altered cells with leucocytes and blood-corpuscles causes the enlargement and swelling of the organ. The epithelial cells lose their striation, the nuclei are obscured, and hyaline droplets often accumulate in them.

(*c*) Interstitial changes. In the milder forms a simple inflammatory exudate—serum mixed with leucocytes and red blood-corpuscles—exists between the tubules. In severer cases areas of small-celled infiltration occur about the capsules and between the convoluted tubes. These changes may be widespread and uniform throughout the organs or more intense in certain regions.

Councilman has described an *acute interstitial nephritis* occurring chiefly in children after fevers, characterized by the presence of cells similar to those described by Unna as plasma cells. He thinks that these cells are formed in other organs, chiefly the spleen and bone marrow, and are carried to the kidneys in the blood current.

Symptoms.—The onset is usually sudden, and when the nephritis follows cold, dropsy may be noticed within twenty-four hours. After fevers the onset is less abrupt, but the patient gradually becomes pale and a puffiness of the face or swelling of the ankles is first noticed. In children there may at the outset be convulsions. Chilliness or rigors initiate the attack in a limited number of cases. Pain in the back, nausea, and vomiting may be present. The fever is variable. Many cases in adults have no rise in temperature. In young children with nephritis from cold or scarlet fever the temperature may, for a few days, range from 101° to 103°.

The most characteristic symptoms are the urinary changes. There may at first be suppression; more commonly the urine is scanty, highly colored, and contains blood, albumin, and tube-casts. The quantity is reduced and only 4 or 5 ounces may be passed in the twenty-four hours; the specific

gravity is high—1.025, or even more; the color varies from a smoky to a deep porter color, but is seldom bright red. On standing there is a heavy deposit; microscopically there are blood-corpuscles, epithelium from the urinary passages, and hyaline, blood, and epithelial tube-casts. The albumin is abundant, forming a curdy, thick precipitate. The total excretion of urea is reduced, though the percentage is high.

Anæmia is an early and marked symptom. In cases of extensive dropsy, effusion may take place into the pleuræ and peritonæum. There are cases of scarlatinal nephritis in which the dropsy of the extremities is trivial and effusion into the pleuræ extensive. The lungs may become œdematous. In rare cases there is œdema of the glottis. Epistaxis may occur or cutaneous ecchymoses may develop in the course of the disease.

The pulse may be hard, the tension increased, and the second sound in the aortic area accentuated. Occasionally dilatation of the heart comes on rapidly and may cause sudden death (Goodhart). The skin is dry and it may be difficult to induce sweating.

Uræmic symptoms develop in a limited number of cases. They may occur at the onset with suppression, more commonly later in the disease. Ocular changes are not so common in acute as in chronic Bright's disease, but hæmorrhagic retinitis may occur and occasionally papillitis.

The course of acute Bright's disease varies considerably. The description just given is of the form which most commonly follows cold or scarlet fever. In many of the febrile cases dropsy is not a prominent symptom, and the diagnosis rests rather with the examination of the urine. Moreover, the condition may be transient and less serious. In other cases, as in the acute nephritis of typhoid fever, there may be hæmaturia and pronounced signs of interference with the renal function. The most intense acute nephritis may exist without anasarca.

In scarlatinal nephritis, in which the glomeruli are most seriously affected, suppression of the urine may be an early symptom, the dropsy is apt to be extreme, and uræmic manifestations are common. Acute Bright's disease in children, however, may set in very insidiously and be associated with transient or slight œdema, and the symptoms may point rather to affection of the digestive system or to brain-disease.

Diagnosis.—It is very important to bear in mind that the most serious involvement of the kidneys may be manifested only by slight œdema of the feet or puffiness of the eyelids, without impairment of the general health. The first indication of trouble may be a uræmic convulsion. This is particularly the case in the acute nephritis of pregnancy, and it is a good rule for the practitioner, when engaged to attend a case, invariably to ask that during the seventh and eighth months the urine should occasionally be sent for examination.

In nephritis from cold and in scarlet fever the symptoms are usually marked and the diagnosis is rarely in doubt. As already mentioned, every case in which albumin is present must not be called acute Bright's disease, not even if tube-casts be present. Thus the common febrile albuminuria, although it represents the first link in the chain of events leading to acute Bright's disease, should not be placed in the same category.

There are occasional cases of acute Bright's disease with anasarca, in which albumin is either absent or present only as a trace. This is a rare condition. Tube-casts are usually found, and the absence of albumin is rarely permanent. The urine may be reduced in amount.

The character of the casts is of use in the diagnosis of the form of Bright's disease, but scarcely of such extreme value as has been stated. Thus, the hyaline and granular casts are common to all varieties. The blood and epithelial casts, particularly those made up of leucocytes, are most common in the acute cases.

Prognosis.—The outlook varies somewhat with the cause of the disease. Recoveries in the form following exposure to cold are much more frequent than after scarlatinal nephritis. In young children the mortality is high, amounting to at least one third of the cases. Serious symptoms are low arterial tension, the occurrence of uræmia, and effusion into the serous sacs. The persistence of the dropsy after the first month, intense pallor, and a large amount of albumin indicate the possibility of the disease becoming chronic. For some months after the disappearance of the dropsy there may be traces of albumin and a few tube-casts.

In a week or ten days, in a case of scarlatinal nephritis, if the progress is favorable, the dropsy diminishes, the urine increases, the albumin lessens, and by the end of a month the dropsy has disappeared and the urine is nearly free. In very young children the course may be rapid, and I have known the urine to be free from albumin in the fourth week. Other cases are more insidious, and though the dropsy may disappear, the albumin persists in the urine, the anæmia is marked, and the condition becomes chronic, or, after several recurrences of the dropsy, improves and complete recovery takes place.

Treatment.—The patient should be in bed and there remain until all traces of the disease have disappeared. As sweating plays such an important part in the treatment, it is well, if possible, to accustom the patient to blankets. He should also be clad in thin Canton flannel.

The diet should consist of milk or butter-milk, gruels made of arrow-root or oat-meal, barley water, and, if necessary, beef tea and chicken broth. It is better, if possible, to confine the patient to a strictly milk diet. As convalescence is established, bread and butter, lettuce, water-cress, grapes, oranges, and other fruits may be given. The return to a meat diet should be gradual.

The patient should drink freely of alkaline mineral waters, ordinary water, or lemonade. The fluids keep the kidneys flushed and wash out the *débris* from the tubes. A useful drink is a drachm of cream of tartar in a pint of boiling water, to which may be added the juice of half a lemon and a little sugar. Taken when cold, this is a pleasant and satisfactory diluent drink.

No remedies, so far as known, control directly the changes which are going on in the kidneys. The indications are: (1) To give the excretory function of the kidney rest by utilizing the skin and the bowels, in the hope that the natural processes may be sufficient to effect a cure; (2) to meet the symptoms as they arise.

In a case of scarlet fever it may occasionally be possible to avert an attack, the premonitory symptoms of which are marked increase in the arterial tension and the presence of blood coloring matter in the urine (Mahomed). An active saline cathartic may completely relieve this condition.

At the onset, when there is pain in the back or hæmaturia, the Paquelin cautery or the dry or wet cups give relief. The last should not be used in children. Warm poultices are often grateful. In cases which set in with suppression of urine, these measures should be adopted, and in addition the hot bath with subsequent pack, copious diluents, and a free purge. The dropsy is best treated by hydrotherapy—either the hot bath, the wet pack, or the hot-air bath. In children the wet pack is usually satisfactory. It is applied by wringing a blanket out of hot water, wrapping the child in it, covering this with a dry blanket, and then with a rubber cloth. In this the child may remain for an hour. It may be repeated daily. In the case of adults, the hot-air bath or the vapor bath may be conveniently given by allowing the vapor or air to pass from a funnel beneath the bed-clothes, which are raised on a low cradle. More efficient, as a rule, is a hot bath of from fifteen or twenty minutes, after which the patient is wrapped in blankets. The sweating produced by these measures is usually profuse, rarely exhausting, and in a majority of cases the dropsy can in this way be relieved. There are some cases, however, in which the skin does not respond to the baths, and if the symptoms are serious, particularly if uræmia supervenes, jaborandi or its active principle, pilocarpine, may be used. The latter may be given hypodermically, in doses of from a sixth to an eighth of a grain in adults, and from a twentieth to a twelfth of a grain in children from two to ten years.

The bowels should be kept open by a morning saline purge; in children the fluid magnesia is readily taken; in adults the sulphate of magnesia may be given by Hay's method, in concentrated form, in the morning, before anything is taken into the stomach. In Bright's disease it not infrequently causes vomiting. The compound powder of jalap, in half-drachm doses, or, if necessary, elaterium may be used. If the dropsy is not extreme, the urine not very concentrated, and uræmic symptoms are not present, the bowels should be kept loose without active purgation. If these measures fail to reduce the dropsy and it has become extreme, the skin may be punctured with a lancet or drained by a small silver canula (Southey's tube), which is inserted beneath it. A fine aspirator needle may be used, and the fluid allowed to drain through a piece of long, narrow rubber tubing into a vessel beneath the bed. If the dyspnœa is marked, owing to pressure of fluid in the pleuræ, aspiration should be performed. In rare instances the ascites is extreme and may require paracentesis, or a Southey's tube may be inserted and the fluid gradually withdrawn. If uræmic convulsions occur, the intensity of the paroxysms may be limited by the use of chloroform; to an adult a pilocarpine injection should be at once given, and from a robust, strong man 20 ounces of blood may be withdrawn. In children the loins may be dry cupped, the wet pack used, and a brisk purgative given. Bromide of potassium and chloral sometimes prove useful.

Vomiting may be relieved by ice and by restricting the amount of food. Drop doses of creasote, iodine, and carbolic acid may be given. The dilute hydrocyanic acid with bismuth is often effectual.

The question of the use of diuretics in acute Bright's disease is not yet settled. The best diuretic, after all, is water, which may be taken freely with the citrate of potash or the benzoate of soda, salts which are held to favor the conversion of the urates into less irritating and more easily excreted compounds. Digitalis and strophanthus are useful diuretics, and may be employed without risk when the arterial tension is low and the cardiac impulse is not forcible. I have never seen any injurious effects from their employment after the early symptoms had lessened in intensity.

For the persistent albuminuria, I agree with Roberts and Rosenstein that we have no remedy of the slightest value. Nothing indicates more clearly our helplessness in controlling kidney metabolism than inability to meet this common symptom. Astringents, alkalies, nitroglycerin, and mercury have been recommended.

For the anæmia always associated with acute Bright's disease iron should be employed. It should not be given until the acute symptoms have subsided. In the adult it may be used in the form of the perchloride in increasing doses, as convalescence proceeds. In children, the syrup of the iodide of iron or the syrup of the phosphate of iron are better preparations. Tyson has recently urged caution in the too free use of iron in kidney disease. The dilatation of the heart is best treated with digitalis, strophanthus, and strychnia.

In the convalescence from acute Bright's disease, care should be taken to guard the patient against cold. The diet should still consist chiefly of milk and a return to mixed food should be gradual. A change of air is often beneficial, particularly a residence in a warm, equable climate.

VII. CHRONIC BRIGHT'S DISEASE.

Here, too, in all forms we deal with a diffuse process, involving epithelial, interstitial, and glomerular tissues. Clinically two groups are recognized—(*a*) the chronic parenchymatous nephritis, which follows the acute attack or comes on insidiously, is characterized by marked dropsy, and post mortem by the *large white kidney.* In the later stages of this process the kidney may be smaller—a condition known as the *small white kidney;* (*b*) chronic interstitial nephritis, in which dropsy is not common and the cardiovascular changes are pronounced. Delafield recognizes a chronic diffuse nephritis with exudation and a chronic productive diffuse nephritis without exudation, the latter corresponding to the contracted kidney of authors.

The amyloid kidney is usually spoken of as a variety of Bright's disease, but in reality it is a degeneration which may accompany any form of nephritis.

Chronic Parenchymatous Nephritis

(Chronic Desquamative and Chronic Tubal Nephritis; Chronic Diffuse Nephritis with Exudation).

Etiology.—In many cases the disease follows the acute nephritis of cold, scarlet fever, or pregnancy. More frequently than is usually stated the disease has an insidious onset and occurs independently of any acute attack. The fevers may play an important *rôle* in certain of these cases. Rosenstein, Bartels, and, in this country, I. E. Atkinson and Thayer have laid special stress upon malaria as a cause. Beer and alcohol are believed to lead to this form of nephritis. In chronic suppuration, syphilis, and tuberculosis the diffuse parenchymatous nephritis is not uncommon, and is usually associated with amyloid disease. Males are rather more subject to the affection than females. It is met with most commonly in young adults, and is by no means infrequent in children as a sequence of scarlatinal nephritis.

Morbid Anatomy.—Several varieties of this form have been recognized. The most common is the *large white kidney* of Wilks, in which the organ is enlarged, the capsule is thin, and the surface white with the stellate veins injected. On section the cortex is swollen and yellowish white in color, and often presents opaque areas. The pyramids may be deeply congested. On microscopical examination it is seen that the epithelium is granular and fatty, and the tubules of the cortex are distended, and contain tube-casts. Hyaline changes are also present in the epithelial cells. The glomeruli are large, the capsules thickened, the capillaries show hyaline changes, and the epithelium of the tuft and of the capsule is extensively altered. The interstitial tissue is everywhere increased, though not to an extreme degree.

The second variety of this form results from the gradual increase in the connective tissue and the subsequent shrinkage, forming what is called the *small white kidney* or the pale granular kidney. It is doubtful whether this is always preceded by the large white kidney. Some observers hold that it may be a primary independent form. The capsule is thickened and the surface is rough and granular. On section the resistance is greatly increased, the cortex is reduced and presents numerous opaque white or whitish-yellow foci, consisting of accumulations of fatty epithelium in the convoluted tubules. This combination of contracted kidney with the areas of marked fatty degeneration has given the name of small granular, fatty kidney to this form. The interstitial changes are marked, many of the glomeruli are destroyed, the degeneration of epithelium in the convoluted tubules is widespread, and the arteries are greatly thickened.

Belonging to this chronic tubal nephritis is a variety known as the *chronic hæmorrhagic nephritis*, in which the organs are enlarged, yellowish white in color, and in the cortex are many brownish-red areas, due to hæmorrhage into and about the tubes. In other respects the changes are identical with those in the large white kidney.

Of changes in the other organs the most marked are thickening of the blood-vessels and hypertrophy of the left heart.

Symptoms.—Following an acute nephritis, the disease may present, in a modified way, the symptoms of that affection. In many cases it sets in insidiously, and after an attack of dyspepsia or a period of failing health and loss of strength the patient becomes pale, and puffiness of the eyelids or swollen feet are noticed in the morning.

The symptoms are as follows: The urine is, as a rule, diminished in quantity, often scanty. It has a dirty-yellow, sometimes smoky, color, and is turbid from the presence of urates. On standing, a heavy sediment falls, in which are found numerous tube-casts of various forms and sizes, hyaline, both large and small, epithelial, granular, and fatty casts. Leucocytes are abundant; red blood-corpuscles are frequently met with, and epithelium from the kidneys and pelves. The albumin is abundant and may amount to one half or one third of the urine boiled. It is more abundant in the urine passed during the day. The specific gravity may be high in the early stages—from 1.020 to 1.025—though in the later stages it is lower. The urea is always reduced in quantity.

Dropsy is a marked and obstinate symptom of this form of Bright's disease. The face is pale and puffy, and in the morning the eyelids are œdematous. The anasarca is general, and there may be involvement of the serous sacs. In these chronic cases associated with large white kidney there is often a distinctive appearance in the face; the complexion is pasty, the pallor marked, and the eyelids are œdematous. The dropsy is peculiarly obstinate. Uræmic symptoms are common, though convulsions are perhaps less frequent than in the interstitial nephritis.

The tension of the pulse is usually increased; the vessels ultimately become stiff and the heart hypertrophied, though there are instances of this form of nephritis in which the heart is not enlarged. The aortic second sound is accentuated. Retinal changes though less frequent than in the chronic interstitial nephritis, occur in a considerable number of cases.

Gastro-intestinal symptoms are common. Vomiting is frequently a distressing and serious symptom, and diarrhœa may be profuse. Ulceration of the colon may occur and prove fatal.

It is sometimes impossible to determine, even by the most careful examination of the urine or by analysis of the symptoms, whether the condition of the kidney is that of the large white or of the small white form. In cases, however, which have lasted for several years, with the progressive increase in the renal connective tissue and the cardio-vascular changes, the clinical picture may approach, in certain respects, that of the contracted kidney. The urine is increased, with low specific gravity. It is often turbid, may contain traces of blood, the tube-casts are numerous and of every variety of form and size, and the albumin is abundant. Dropsy is usually present, though not so extensive as in the early stages.

The *prognosis* is extremely grave. In a case which has persisted for more than a year recovery rarely takes place. Death is caused either by great effusion with œdema of the lungs, by uræmia, or by secondary inflammation of the serous membranes. Occasionally in children, even when the disease has persisted for two years, the symptoms disappear and recovery takes place.

Treatment.—Essentially the same treatment should be carried out as in acute Bright's disease. Milk or butter-milk should constitute the chief article of food. The dropsy should be treated by hydrotherapy. Iron preparations should be given when there is marked anæmia. It is to be remembered that the pallor of the face may not be a good index of the blood condition. Tyson thinks that the profession has been much too free in the use of iron in these cases. The acetate of potash, digitalis, and diuretin are useful in increasing the flow of urine. Basham's mixture given in plenty of water will be found beneficial.

Chronic Interstitial Nephritis

(Contracted Kidney; Granular Kidney; Cirrhosis of the Kidney; Gouty Kidney; Renal Sclerosis).

Sclerosis of the kidney is met with (*a*) as a sequence of the large white kidney, forming the so-called pale granular or secondary contracted kidney; (*b*) as an independent primary affection; (*c*) as a sequence of arteriosclerosis.

Etiology.—The primary form is chronic from the outset, and is a slow, creeping degeneration of the kidney substance—in many respects only an anticipation of the gradual changes which take place in the organ in extreme old age. In many cases no satisfactory cause can be assigned. In others there are hereditary influences, as in the remarkable family studied by Dickinson, in which a pronounced tendency to chronic Bright's disease occurred in four generations. Families in which the arteries tend to degenerate early are more prone to interstitial nephritis. Syphilis is held by some to be a cause. Alcohol probably plays an important part, particularly in conjunction with other factors. Among the better classes in this country chronic Bright's disease is very common, and is, I believe, caused more frequently by overeating than by excesses in alcohol. Some believe excessive use of meat is injurious, since it increases the materials out of which uric acid is formed. By many a functional disorder of the liver, leading to lithæmia, is regarded as the most efficient factor. It is quite possible that in persons who habitually eat and drink too much the work thrown upon this organ is excessive, and the elaboration of certain materials is so defective that in their excretion from the general circulation they irritate the kidneys.

Actual gout, which in England is a common cause of interstitial nephritis, is not an important factor here. On the other hand, the nutritional disorder known as lithæmia is very common, either with or without dyspepsia. Lead, as is well known, may produce renal sclerosis, but it is a minor factor in comparison with other causes. It is doubtful if climate has any influence. Purdy regards the cold, moist regions of the Northeastern States as specially favorable to the disease.

Other factors which may account for the prevalence of chronic Bright's disease in the better classes in this country may be the intense worry and strain of business, combined, as they often are, with habits of hurried and excessive eating and a lack of proper exercise. Males are more commonly

attacked than females. Under twenty-five years of age it is a rare disease; between twenty-five and forty a few well-marked cases occur; between forty and sixty it is common.

Morbid Anatomy.—The kidneys are usually small, and together may weigh no more than an ounce and a half. The capsule is thick and adherent; the surface of the organ irregular and covered with small nodules, which have given to it the name of granular kidney. In stripping off the capsule, portions of the kidney substance are removed. Small cysts are frequently seen on the surface. The color is usually reddish, often a very dark red. On section the substance is tough and resists cutting; the cortex is thin and may measure no more than a couple of millimetres. The pyramids are less wasted. The small arteries are greatly thickened and stand out prominently. The fat about the pelvis is greatly increased.

Microscopically there is seen a marked increase in the connective tissue and degeneration and atrophy of the secreting structures, glomerular and tubal, the former predominating and giving the main characters to the lesion. The following are the most important changes:

(*a*) An increase in the fibrous elements, widely distributed throughout the organ, but more advanced in the cortex, particularly in the tissue between the medullary rays. In the pyramids the distribution of new growth is less patchy and more diffuse. In the early stages of the process there is a small-celled infiltration between the tubes and around the glomeruli, and finally this becomes fibrillated and is seen encircling the tubules and Bowman's capsules, around the latter often forming concentric layers.

(*b*) The changes in the glomeruli are striking, and in advanced cases a very considerable number of them have undergone complete atrophy and are represented as densely encapsulated hyaline structures. The atrophy is partly due to changes in the capillary walls and multiplication of cells between the loops, partly to extensive hyaline degeneration, and in part, no doubt, to the alterations in the afferent vessels. The normal glomeruli usually show some thickening of the capsule and increase in the cells of the tufts.

(*c*) The tubules show changes in the epithelium, which vary a good deal in different localities. Where the connective-tissue growth is most advanced they are greatly atrophied and the epithelium may be represented by small cubical cells. In other instances the epithelium has entirely disappeared. On the other hand, in the regions represented by the projecting granules the tubules are usually dilated, and the epithelium shows hyaline, fatty, and granular changes. Very many of them contain dark masses of epithelial *débris* and tube-casts. In the interstitial tissue and in the tubules there may be pigmentary changes due to hæmorrhage. The dilatation of the tubules may reach an extreme grade, forming definite cysts.

(*d*) The arteries show an advanced sclerosis. The intima is greatly thickened and there are changes in the adventitia and in the media, consisting in increase in the thickness due to proliferation of the connective tissue, in the latter coat at the expense of the muscular elements.

The view most generally entertained at present is that the essential lesion is in the secreting tissues of the tubules and the glomeruli, and that

the connective-tissue overgrowth is secondary to this. Greenfield holds that the primary change is in most instances in the glomeruli, to which both the degeneration in the epithelium of the convoluted tubules and the increase in the intertubular connective tissue are secondary.

Associated with contracted kidney are general arterio-sclerosis and hypertrophy of the heart. The changes in the arteries have already been described in the section on arterio-sclerosis. The hypertrophy of the heart is constant, and the enlargement may reach an extreme grade. Variations depend, no doubt, in part upon the extent of the diffuse arterial degeneration, but there are instances in which the term *cor bovinum* may be applied to the enlarged organ. In such cases the hypertrophy is not confined to the left ventricle, but involves the entire heart. The explanation of this hypertrophy has been much discussed. It was at first held to be due to the increased work thrown upon the organ in driving the impure blood through the capillary system. Basing his opinion upon the supposed muscular increase in the smaller arteries, Johnson regarded the hypertrophy as an effort to overcome a sort of stop-cock action of these vessels, which, under the influence of the irritating ingredient in the blood, contracted and increased greatly the peripheral resistance. Traube believed that the obliteration of a large number of capillary territories in the kidney materially raised the arterial pressure, and in this way led to the hypertrophy of the heart; an additional factor, he thought, was the diminished excretion of water, which also heightened the pressure within the blood-vessels.

With our present knowledge the most satisfactory explanation is that given by Cohnheim, which is thus clearly and succinctly put by Fagge: "He gives reasons for thinking that the activity of the circulation through the kidneys at any moment—in other words, the state of the smaller renal arteries as regards contraction or dilatation—depends not (as in the case of the tissues generally) upon the need of those organs for blood, but solely upon the amount of material for the urinary secretion that the circulatory fluid happens then to contain. This suggestion has bearings . . . upon the development of hypertrophy in one kidney when the other has been entirely destroyed. But another consequence deducible from it is that when parts of both kidneys have undergone atrophy, the blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact. But in order that such a quantity of blood should pass through the restricted capillary area now open to it, an excessive pressure must obviously be necessary. This can be brought to bear only by the exertion of more than the normal degree of force on the part of the left ventricle, combined with the maintenance of a corresponding resistance in all other districts of the arterial system. And so one can account at once for the high arterial pressure and for the cardio-vascular changes that are secondary to it."

Symptoms.—Perhaps a majority of the cases are latent, and are not recognized until the occurrence of one of the serious or fatal complications. Even an advanced grade of contracted kidney may be compatible with great mental and bodily activity. There may have been no symptoms whatever to suggest to the patient the existence of a serious malady. In other cases

the general health is disturbed. The patient complains of lassitude, is sleepless, has to get up at night to micturate; the digestion is disordered, the tongue is furred; there are complaints of headache, failing vision, and breathlessness on exertion.

So complex and varied is the clinical picture of chronic Bright's disease that it will be best to consider the symptoms under the various systems.

Urinary System.—The amount of urine is usually increased, and from 2 to 4 litres may be passed. Frequently the patient has to get up two or three times during the night to empty the bladder, and there is increased thirst. It is for these symptoms occasionally that relief is sought. It is to be remembered, however, that frequent micturition at night may be associated with irritability of the prostate and, in certain cases, with superacidity of the urine. The secretion is clear, the mucous cloud is well marked, but there is no definite sediment. The color is a light yellow, and the specific gravity ranges from 1.005 to 1.012. Persistent low specific gravity is one of the most constant and important features of the disease. Traces of albumin are found, but may be absent at times, particularly in the early morning urine. It is often simply a slight cloudiness, and may be apparent only with the more delicate tests. The sediment is scanty, and in it a few hyaline or granular casts are found. The quantity of the solid constituents of the urine is, as a rule, diminished, though in some instances the urea may be excreted in full amount. In attacks of dyspepsia or bronchitis, or in the later stages when the heart fails, the quantity of albumin may be greatly increased and the urine diminished. Occasionally blood occurs in the urine, and there may even be hæmaturia (S. West). Slight leakage, represented by the constant presence of a few red cells, may be present early in the disease and persist for years. In other instances there may be, particularly after exercise, flecks of blood in a pale, smoky urine.

Circulatory System.—The pulse is hard, the tension increased, and the vessel wall, as a rule, thickened. As already mentioned, a distinction must be made between increased tension and thickening of the arterial wall. The tension may be plus in a normal vessel, but in chronic Bright's disease it is more common to have increased tension in a stiff artery.

A pulse of increased tension has the following characters: It is hard and incompressible, requiring a good deal of force to overcome it; it is persistent, and in the intervals between the beats the vessel feels full and can be rolled beneath the finger. These characters may be present in a vessel the walls of which are little, if at all, increased in thickness. To estimate the latter the pulse wave should be obliterated in the radial, and the vessel wall felt beyond it. In a perfectly normal vessel the arterial coats, under these circumstances, cannot be differentiated from the surrounding tissue; whereas, if thickened, the vessel can be rolled beneath the finger. Persistent high tension is one of the earliest and most important symptoms of interstitial nephritis. The cardiac features are equally important, though often less obvious. Hypertrophy of the left ventricle occurs to overcome the resistance offered in the arteries. The enlargement of the heart ultimately becomes more general. The apex is displaced downward and to the left; the impulse is forcible and may be heaving. In elderly persons with

In other parts of the eye, as in the conjunctivae and eyelids, there may be recurring haemorrhages, which are sometimes [illegible] (de Schweinitz)

emphysema, the displacement of the apex may not be evident. The first sound at the apex may be duplicated; more commonly the second sound at the aortic cartilage is accentuated, a very characteristic sign of increased tension. The sound in extreme cases may have a bell-like quality. In many cases a systolic murmur develops at the apex, probably as a result of relative insufficiency. It may be loud and transmitted to the axilla. Finally the hypertrophy fails, the heart becomes dilated, gallop rhythm is present, and the general condition is that of a chronic heart-lesion.

Respiratory System.—Sudden œdema of the glottis may occur. Effusion into the pleuræ or sudden œdema of the lungs may prove fatal. Acute pleurisy and pneumonia are not uncommon. Bronchitis is a frequent accompaniment, particularly in the winter. Sudden attacks of oppressed breathing, particularly at night, are not infrequent. This is often a uræmic symptom, but is sometimes cardiac. The patient may sit up in bed and gasp for breath, as in true asthma. Cheyne-Stokes breathing may be present, most commonly toward the close, but the patient may be walking about and even attending to his occupation.

Digestive System.—Dyspepsia and loss of appetite are common. Severe and uncontrollable vomiting may be the first symptom. This is usually regarded as a manifestation of uræmia, but it may be present without any other indications, and I have known it to prove fatal without any suspicion that chronic Bright's disease was present. Severe and even fatal diarrhœa may develop. The tongue may be coated and the breath heavy and urinous.

Nervous System.—Various cerebral manifestations have already been mentioned under uræmia. Headache, sometimes of the migraine type, may be an early and persistent feature of chronic Bright's disease. Cerebral apoplexy is closely related to interstitial nephritis. The hæmorrhage may take place into the meninges or the cerebrum. It is usually associated with marked changes in the vessels. Neuralgias, in various regions, are not uncommon.

Special Senses.—Troubles in vision may be the first symptom of the disease. It is remarkable in how many cases of interstitial nephritis the condition is diagnosed first by the ophthalmic surgeon. The flame-shaped retinal hæmorrhages are the most common. Less frequent is diffuse retinitis or papillitis. Sudden blindness may supervene without retinal changes—uræmic amaurosis. Diplopia is a rare event. I have seen but one case. Knies says that it is frequent. Auditory troubles are by no means infrequent in chronic Bright's disease. Ringing in the ears, with dizziness, is not uncommon. Various forms of deafness may occur.

Skin.—Œdema is not common in interstitial nephritis. Slight puffiness of the ankles may be present, but in a majority of the cases dropsy does not supervene. When extensive, it is almost always the result of gradual failure of the hypertrophied heart. The skin is often dry and pale, and sweats are not common. In some instances the sweat may deposit a white frost of urea on the surface of the skin. Eczema is a common accompaniment of chronic interstitial nephritis. Tingling of the fingers or numbness and pallor—the dead fingers—are not, as some suppose, in any way

peculiar to Bright's disease. Intolerable itching of the skin may be present, and cramps in the muscles are by no means rare.

Hæmorrhages are not infrequent; thus, epistaxis may occur and prove serious. Purpura may develop. Broncho-pulmonary hæmorrhages are said, by some French writers, to be common, but no instance of it has come under my observation. Ascites is rare except in association with cirrhosis of the liver.

Diagnosis.—The autopsy often discloses the true nature of the disease, one of the many intercurrent affections of which may have proved fatal. The early stages of interstitial nephritis are not recognizable. In a patient with increased pulse tension (particularly if the vessel wall is sclerotic), with the apex beat of the heart dislocated to the left, the second aortic sound ringing and accentuated, the urine abundant and of low specific gravity, with a trace of albumin and an occasional hyaline or granular cast, the diagnosis of interstitial nephritis may be safely made. Of all the indications, that offered by the pulse is the most important. Persistent high tension with thickening of the arterial wall in a man under fifty means that serious mischief has already taken place, that cardio-vascular changes are certainly, and renal most probably, present. It is important in the diagnosis of this condition not to rest content with a single examination of the urine. Both the evening and the morning secretion should be studied. The sediment should be collected in a conical glass, and in looking for tube-casts a large surface should be examined with a tolerably low power and little light. The arterio-sclerotic kidney may exist for a long time without the occurrence of albumin, or the albumin may be in very small quantities. In many cases it is impossible to differentiate the primary interstitial nephritis from an arterio-sclerotic kidney, nor clinically is it of any special value so to do. In persons under forty, with very high tension, great thickening of the superficial arteries, and marked hypertrophy of the heart, the renal are more likely to be secondary to the arterial changes.

Prognosis.—Chronic Bright's disease is an incurable affection, and the anatomical conditions on which it depends are quite as much beyond the reach of medicines as wrinkled skin or gray hair. Interstitial nephritis, however, is compatible with the enjoyment of life for many years, and it is now universally recognized that increased tension, thickening of the arterial walls, and polyuria with a small quantity of albumin, neither doom a man to death within a short time nor necessarily interfere with the pursuits of an active life so long as proper care be taken. I know patients who have had high tension and a little albumin in the urine with hyaline casts for ten, twelve, and, in one instance, fifteen years. Serious indications are the development of uræmic symptoms, dilatation of the heart, the onset of serous effusions, the development of Cheyne-Stokes breathing, persistent vomiting, and diarrhœa.

Treatment.—Patients without local indications or in whom the condition has been accidentally discovered should so regulate their lives as to throw the least possible strain upon heart, arteries, and kidneys. A quiet life without mental worry, with gentle but not excessive exercise, and residence in an equable climate, should be recommended. In addition they

should be told to keep the bowels regular, the skin active by a daily tepid bath with friction, and the urinary secretion free by drinking daily a definite amount of either distilled water or some pleasant mineral water. Alcohol should be strictly prohibited. Tea and coffee are allowable.

The diet should be light and nourishing, and the patient should be warned not to eat excessively, and not to take meat more than once a day. Care in food and drink is probably the most important element in the treatment of these early cases.

A patient in good circumstances may be urged to go away during the winter months, or, if necessary, to move altogether to a warm equable climate, like that of Southern California. There is no doubt of the value in these cases of removal from the changeable, irregular weather which prevails in the temperate regions from November until April.

At this period medicines are not required unless for certain special symptoms. Patients derive much benefit from an annual visit to certain mineral springs, such as Poland, Bedford, Saratoga, in this country, and Vichy and others in Europe. Mineral waters have no curative influence upon chronic Bright's disease; they simply help the interstitial circulation and keep the drains flushed. In this early stage, when the patient's condition is good, the tension not high, and the quantity of albumin small, medicines are not indicated, since no remedies are known to have the slightest influence upon the progress of the disease. Sooner or later symptoms arise which demand treatment. Of these the following are the most important:

(*a*) *Greatly Increased Arterial Tension.*—It is to be remembered that a certain increase of tension is not only necessary but unavoidable in chronic Bright's disease, and probably the most serious danger is too great lowering of the blood tension. The happy medium must be sought between such heightened tension as throws a serious strain upon the heart and risks rupture of the vessels and the low tension which, under these circumstances, is specially liable to be associated with serous effusions. In cases with persistent high tension the diet should be light, an occasional saline purge should be given, and sweating promoted by means of hot air or the hot bath. If these measures do not suffice, nitroglycerin may be tried, beginning with 1 minim of the 1-per-cent solution three times a day, and gradually increasing the dose if necessary. Patients vary so much in susceptibility to this drug that in each case it must be tested, the limit of dosage being that at which the patient experiences the physiological effect. As much as 10 minims of the 1-per-cent solution may be given three times a day. In many case I have given it in much larger doses for weeks at a time. I have never seen any ill effects from it. If the dose is excessive the patients complain at once of flushing or headache. Its use may be kept up for six or seven weeks, then stopped for a week and resumed. Its value is seen not only in the reduction of the tension, but also in the striking manner in which it relieves the headache, dizziness, and dyspnœa.

(*b*) More or less *anæmia* is present in advanced cases, and is best met by the use of iron. Weir Mitchell, who has had a unique experience in certain forms of chronic Bright's disease, gives the tincture of the per-

chloride of iron in large doses—from half a drachm to a drachm three times a day. He thinks that it not only benefits the anæmia, but that it also is an important means of reducing the arterial tension.

(*c*) Many patients with Bright's disease present themselves for treatment with signs of cardiac dilatation; there is a gallop rhythm or the heart sounds have a fœtal character, the breath is short, the urine scanty and highly albuminous, and there are signs of local dropsy. In these cases the treatment must be directed to the heart. A morning dose of salts or calomel may be given, and digitalis in 10-minim doses, three or four times a day. Strychnia may be used with benefit in this condition. In some instances other cardiac tonics may be necessary, but as a rule the digitalis acts promptly and well.

(*d*) *Uræmic Symptoms.*—Even before marked manifestations are present there may be extreme restlessness, mental wandering, a heavy, foul breath, and a coated tongue. Headache is not often complained of, though intense frontal headache may be an early symptom of uræmia. In this condition, too, the patient may complain of palpitation, feelings of numbness, and sometimes nocturnal cramps. For these symptoms the saline purgatives should be ordered, and hot baths, so as to induce copious sweating. Grandin states that irrigation of the bowel with water at a temperature from 120° to 150° is most useful. Nitroglycerin also may be freely used to reduce the tension. For the uræmic convulsions, if severe, inhalations of chloroform may be used. If the patient is robust and full-blooded, from 12 to 20 ounces of blood should be removed. The patient should be freely sweated, and if the convulsions tend to recur chloral may be given, either by the mouth or per rectum, or, better still, morphia. Uræmic coma must be treated by active purgation, and sweating should be promoted by the use of pilocarpine or the hot bath. For the restlessness and delirium morphia is indispensable. Since its recommendation in uræmic states some years ago, by Stephen MacKenzie, I have used this remedy extensively and can speak of its great value in these cases. I have never seen ill effects or any tendency to coma follow. It is of special value in the dyspnœa and Cheyne-Stokes breathing of advanced arterio-sclerosis with chronic uræmia.

VIII. AMYLOID DISEASE.

Amyloid (lardaceous or waxy) degeneration of the kidneys is simply an event in the process of chronic Bright's disease, most commonly in the chronic parenchymatous nephritis following fevers, or of cachectic states. It has no claim to be regarded as one of the varieties of Bright's disease. The affection of the kidneys is generally a part of a widespread amyloid degeneration occurring in prolonged suppuration, as in disease of the bone, in syphilis, tuberculosis, and occasionally leukæmia, lead poisoning, and gout. It varies curiously in frequency in different localities.

Anatomically the amyloid kidney is large and pale, the surface smooth, and the venæ stellatæ well marked. On section the cortex is large and may show a peculiar glistening, infiltrated appearance, and the glomeruli

are very distinct. The pyramids, in striking contrast to the cortex, are of a deep red color. A section soaked in dilute tincture of iodine shows spots of a walnut or mahogany brown color. The Malpighian tufts and the straight vessels may be most affected. In lardaceous disease of the kidneys the organs are not always enlarged. They may be normal in size or small, pale, and granular. The amyloid change is first seen in the Malpighian tufts, and then involves the afferent and efferent vessels and the straight vessels. It may be confined entirely to them. In later stages of the disease the tubules are affected, chiefly the membrane, rarely, if ever, the cells themselves. In addition, the kidneys always show signs of diffuse nephritis. The Bowman's capsules are thickened, there may be glomerulitis, and the tubal epithelium is swollen, granular, and fatty.

Symptoms.—The renal features alone may not indicate the presence of this degeneration. Usually the associated condition gives a hint of the nature of the process. The urine, as a rule, shows important changes; the quantity is increased, and it is pale, clear, and of low specific gravity. The albumin is usually abundant, but it may be scanty, and in rare instances absent. Possibly the variations in the situation of the amyloid changes may account for this, since albumin is less likely to be present when the change is confined to the vasa recta. In addition to ordinary albumin globulin may be present. The tube-casts are variable, usually hyaline, often fatty or finely granular. Occasionally the amyloid reaction can be detected in the hyaline casts. Dropsy is present in many instances, particularly when there is much anæmia or profound cachexia. It is not, however, an invariable symptom, and there are cases in which it does not develop. Diarrhœa is a common accompaniment.

Increased arterial tension and cardiac hypertrophy are not usually present, except in those cases in which amyloid degeneration occurs in the secondary contracted kidney; under which circumstances there may be uræmia and retinal changes, which, as a rule, are not met with in other forms.

Diagnosis.—By the condition of the urine alone it is not possible to recognize amyloid changes in the kidney. Usually, however, there is no difficulty, since the Bright's disease comes on in association with syphilis, prolonged suppuration, disease of the bone, or tuberculosis, and there is evidence of enlargement of the liver and spleen. A suspicious circumstance is the existence of polyuria with a large amount of albumin in the urine, or when, in these constitutional affections, a large quantity of clear, pale urine is passed, even without the presence of albumin.

The prognosis depends rather on the condition with which the nephritis is associated. As a rule it is grave.

The treatment of the condition is that of chronic Bright's disease.

IX. PYELITIS

(Consecutive Nephritis; Pyelonephritis; Pyonephrosis).

Definition.—Inflammation of the pelvis of the kidney and the conditions which result from it.

Etiology.—Pyelitis is induced by many causes, among which the following are the most important: (*a*) The irritation of calculi—a very frequent cause. (*b*) Tubercle. (*c*) The infectious pyelitis which develops in fevers, in which an acute inflammation of the pelvis of the kidney may occur, sometimes hæmorrhagic in character, more frequently diphtheritic. (*d*) The presence of decomposing urine, following pressure upon the ureter by tumors or bladder-disease. By far the most frequent form of pyelitis is that which is consecutive to cystitis, from whatever cause. In these cases the inflammation may not be confined to the pelvis, but pass to the kidney, inducing pyelonephritis. (*e*) Occasional causes are cancer, hydatids, the ova of certain parasites, and, according to some, the irritation of the saccharine urine of diabetes, and the irritation of turpentine or cubebs. (*f*) A primary pyelitis or pyelonephritis has been described as coming on after cold or overexertion, but such cases are extremely rare. The condition is met with in children (Holt), and in one case which I saw with Holmes, of Chatham, the pus and the chills, after recurring at intervals for many months, disappeared after circumcising the boy, who had a very narrow prepuce. (*g*) Following attacks of Dietl's crises in movable kidney pyelitis may be present.

Morbid Anatomy.—In the early stages of pyelitis the mucous membrane is turbid, somewhat swollen, and may show ecchymoses or a grayish pseudo-membrane. The urine in the pelvis is cloudy, and, on examination, numbers of epithelial cells are seen.

In the calculous pyelitis there may be only slight turbidity of the membrane, which has been called by some catarrhal pyelitis. More commonly the mucosa is roughened, grayish in color, and thick. Under these circumstances there is almost always more or less dilatation of the calyces and flattening of the papillæ. Following this condition there may be (*a*) extension of the suppurative process to the kidney itself, forming a pyelonephritis; (*b*) a gradual dilatation of the calyces with atrophy of the kidney substance, and finally the production of the condition of pyonephrosis, in which the entire organ is represented by a sac of pus with or without a thin shell of renal tissue. (*c*) After the kidney structure has been destroyed by suppuration, if the obstruction at the orifice of the pelvis persists, the fluid portions may be absorbed and the pus become inspissated, so that the organ is represented by a series of sacculi containing grayish, putty-like masses, which may become impregnated with lime salts.

Tuberculous pyelitis, as already described, usually starts upon the apices of the pyramids, and may at first be limited in extent. Ultimately the condition produced may be similar to that of calculous pyelitis. Pyonephrosis is quite as frequent a sequence, while the final transformation of

the pus into a putty-like material impregnated with salts, forming the so-called scrofulous kidney, is even commoner.

The pyelitis consecutive to cystitis is usually bilateral, and the kidney is apt to be involved, forming the so-called *surgical kidney*—acute suppurative nephritis. There are lines of suppuration extending along the pyramids, or small abscesses in the cortex, often just beneath the capsule; or there may be wedge-shaped abscesses. The pus organisms either pass up the tubules or, as Steven has shown, through the lymphatics.

Symptoms.—The forms associated with the fevers rarely cause any symptoms, even when the process is extensive. In mild grades there is pain in the back or there may be tenderness on deep pressure on the affected side. The urine is turbid, contains a few mucous and pus cells, and occasionally blood-corpuscles. The urine is acid, and there may be a trace of albumin.

Before the condition of pyuria is established there may be attacks of pain on the affected side (not amounting to the severe agony of renal colic), rigors, high fever, and sweats. Under these circumstances the urine, which may have been clear, becomes turbid or smoky from the presence of blood, and may contain large numbers of mucus cells and transitional epithelium. These cases are not common, but I have twice had opportunity of studying such attacks for a prolonged period. In one patient the occurrence of the rigor and fever could sometimes be predicted from the change in the condition of the urine. Such cases occur, I believe, in association with calculi in the pelvis.

The statement is not infrequently made that the epithelium in the urine in pyelitis is distinctive and characteristic. This is erroneous, as may be readily demonstrated by comparing scrapings of the mucosa of the renal pelvis and of the bladder. In both the epithelium belongs to what is called the transitional variety, and in both regions the same conical, fusiform and irregular cells with long tails are found.

When the pyelitis, whether calculous or tuberculous, has become chronic and discharges, the symptoms are:

(1) *Pyuria.*—The pus is in variable amount, and may be intermittent. Thus, as is often the case when only one kidney is involved, the ureter may be temporarily blocked, and normal urine is passed for a time; then there is a sudden outflow of the pent-up pus and the urine becomes purulent. Coincident with this retention, a tumor mass may be felt on the side affected. The pus has the ordinary characters, but the transitional epithelium is not so abundant at this stage and comes from the bladder or from the pelvis of the healthy side. Occasionally in rapidly advancing pyelonephritis, portions of the kidney tissue, particularly of the apices of the pyramids, may slough away and appear in the urine; or, as in a remarkable specimen shown to me by Tyson, solid cheesy moulds of the calyces are passed. Casts from the kidney tubules are sometimes present. The reaction of the urine is at first acid, and may remain so even when the pus is passed in large quantities. If it remains any time in the bladder or if cystitis exists it becomes ammoniacal. Micturition may be very frequent and irritability of the bladder may be present.

(2) Intermittent fever associated with rigors is usually present in cases of suppurative pyelitis. The chills may recur at regular intervals, and the cases are often mistaken for malaria. Owen-Rees called attention to the frequent occurrence of these rigors, which form a characteristic feature of both calculous and tuberculous pyelitis. Ultimately the fever assumes a hectic type and the rigors may cease.

(3) The general condition of the patient usually indicates prolonged suppuration. There is more or less wasting with anæmia and a progressive failure of health. Secondary abscesses may develop and the clinical picture becomes that of pyæmia. In some instances, particularly of tuberculous pyelitis, the clinical course may resemble that of typhoid fever. There are instances of pyuria recurring, at intervals, for many years without impairment of the bodily vigor.

(4) Physical examination in chronic pyclitis usually reveals tenderness on the affected side or a definite swelling, which may vary much in size and ultimately attain large dimensions if the kidney becomes enormously distended, as in pyonephrosis.

(5) Occasionally nervous symptoms, which may be associated with dyspnœa, supervene, or the termination may be by coma, not unlike that of diabetes. These have been attributed to the absorption of the decomposing materials in the urine, whence the so-called ammoniæmia. A form of paraplegia has been described in connection with some cases of abscess of the kidney, but whether due to a myelitis or to a peripheral neuritis has not yet been determined.

In suppurative nephritis or surgical kidney following cystitis, the patient complains of pain in the back, the fever becomes high, irregular, and associated with chills, and in acute cases a typhoid state develops in which death occurs.

Diagnosis.—Between the tuberculous and the calculous forms of pyelitis it may be difficult or impossible to distinguish, except by the detection of tubercle bacilli in the pus. The examination for bacilli should be made systematically in all suspicious cases. The tuberculin test may be used with advantage. From perinephric abscess pyonephrosis is distinguished by the more definite character of the tumor, the absence of œdematous swelling in the lumbar region, and, most important of all, the history of the case. The urine, too, in perinephric abscess may be free from pus. There are cases, however, in which it is difficult indeed to make a satisfactory diagnosis. A patient, whom I saw with Fussell, had had cystitis through her pregnancy, subsequently pus in the urine for several months, and then a large fluctuating abscess developed in the right lumbar region. It did not seem possible, either before or during the operation, to determine whether the case was a simple pyonephrosis or whether there had been a perinephric abscess caused by the pyelitis.

Suppurative pyelitis and cystitis are frequently confounded. I have known instances of the former in which perineal section was performed on the supposition of the existence of an intractable cystitis. The two conditions may, of course, coexist and prove puzzling, but the history, the acid character of the pus in many instances, the less frequent occurrence of am-

moniacal decomposition, the local signs in one lumbar region, and the absence of pain in the bladder should be sufficient to differentiate the affections. In women, by catheterization of the ureters, it may be definitely determined whether the pus comes from the kidneys or from the bladder. The cystoscope may be used for this purpose.

Prognosis.—Cases coming on during the fevers usually recover. Tuberculous pyelitis may terminate favorably by inspissation of the pus and conversion into a putty-like substance with deposition of lime salts. When pyonephrosis develops the dangers are increased. Perforation may occur, the patient may be worn out by the hectic fever, or amyloid disease may develop.

Treatment.—In mild cases fluids should be taken freely, particularly the alkaline mineral waters, to which the citrate of potash may be added.

The treatment of the calculous form will be considered later. Practically there are no remedies which have much influence upon the pyuria. Astringents in no way control the discharge, nor have I seen the slightest benefit from buchu, copaiba, sandal-wood oil, or uva ursi. Tonics should be given, a nourishing diet, and milk and butter-milk may be taken freely. When the tumor has formed or even before it is perceptible, if the symptoms are serious and severe, the kidney should be explored, and, if necessary, nephrotomy should be performed.

X. HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calyces of the kidney with atrophy of its substance, caused by the accumulation of non-purulent fluids, the result of obstruction.

Etiology.—The condition may be congenital, owing to some abnormality in the ureter or urethra. The tumor produced may be large enough to retard labor. Sometimes it is associated with other malformations. There is a condition of moderate dilatation, apparently congenital, which is not connected with any obstruction in the ducts. A case of the kind was shown at the Philadelphia Pathological Society by Daland.

In some instances there has been contraction or twisting of the ureter, or it has been inserted into the kidney at an acute angle or at a high level. In adult life the condition may be due to lodgment of a calculus, or to a cicatricial stricture following ulcer.

New growths, such as tubercle or cancer, occasionally induce hydronephrosis; more commonly, pressure upon the ureter from without, particularly tumors of the ovaries and uterus. Occasionally cicatricial bands compress the ureter. Obstruction within the bladder may result from cancer, from hypertrophy of the prostate with cystitis, and in the urethra from stricture. It is stated that slight grades of hydronephrosis have been found in patients with excessive polyuria.

In whatever way produced, when the ureter is blocked the secretion accumulates in the pelvis and infundibula. Sometimes acute inflammation follows, but more commonly the slow, gradual pressure causes atrophy of

the papillæ with gradual distention and wasting of the organ. In acquired cases from pressure, even when dilatation is extreme, there may usually be seen a thin layer of renal structure. In the most extreme stages the kidney is represented by a large cyst, which may perhaps show on its inner surface imperfect septa. The fluid is thin and yellowish in color, and contains traces of urinary salts, urea, uric acid, and sometimes albumin. The secretion may be turbid from admixture with small quantities of pus.

Total occlusion does not always lead to a hydronephrosis, but may be followed by atrophy of the kidney. It appears that when the obstruction is intermittent or not complete the greatest dilatation is apt to follow. The sac may be enormous, and cause an abdominal tumor of the largest size. The condition has even been mistaken for ascites. Enlargement of the other kidney may compensate for the defect. Hypertrophy of the left side of the heart usually follows.

Symptoms.—When small, it may not be noticed. The congenital cases when bilateral usually prove fatal within a few days; when unilateral, the tumor may not be noticed for some time. It increases progressively and has all the characters of a tumor in the renal region. In adult life many of the cases, due to pressure by tumors, as in cancer of the uterus and enlargement of the prostate, etc., give rise to no symptoms.

There are remarkable instances of *intermittent* hydronephrosis in which the tumor suddenly disappears with the discharge of a large quantity of clear fluid. The sac gradually refills, and the process may be repeated for years. In these cases the obstruction is unilateral; a cicatricial stricture exists, or a valve is present in the ureter, or the ureter enters the upper part of the pelvis. Many of the cases are in women and associated with movable kidney.

The examination of the abdomen shows, in unilateral hydronephrosis, a tumor occupying the renal region. When of moderate size it is readily recognized, but when large it may be confounded with ovarian or other tumors. In young children it may be mistaken for sarcoma of the kidney or of the retroperitoneal glands, the common cause of abdominal tumor in early life. Aspiration alone would enable us to differentiate between hydronephrosis and tumor. The large hydronephrotic sac is frequently mistaken for ovarian tumor. The latter is, as a rule, more mobile, and rarely fills the deeper portion of the lumbar region so thoroughly. The ascending colon can often be detected passing over the renal tumor, and examination per vaginam, particularly under ether, will give important indications as to the condition of the ovaries. In doubtful cases the sac should be aspirated. The fluid of the renal cyst is clear, or turbid from the presence of cell elements, rarely colloid in character; the specific gravity is low; albumin and traces of urea and uric acid are usually present; and the epithelial elements in it may be similar to those found in the pelvis of the kidney. In old sacs, however, the fluid may not be characteristic, since the urinary salts disappear, but in one case of several years' duration oxalate of lime and urea were found.

Perhaps the greatest difficulty is offered by the condition of hydronephrosis in a movable kidney. Here, the history of sudden disappear-

ance of the tumor with the passage of a large quantity of clear fluid would be a point of great importance in the diagnosis. In those rare instances of an enormous sac filling the entire abdomen, and sometimes mistaken for ascites, the character of the fluid might be the only point of difference. The tumor of pyonephrosis may be practically the same in physical characteristics. Fever is usually present, and pus is often found in the urine. In these cases, when in doubt, exploratory puncture should be made.

The outlook in hydronephrosis depends much upon the cause. When single, the condition may never produce serious trouble, and the intermittent cases may persist for years and finally disappear. Occasionally the cyst ruptures into the peritonæum, more rarely through the diaphragm into the lung. A remarkable case of this kind was under the care of my colleague, Halsted. A man, aged twenty-one, had, from his second year, attacks of abdominal pain in which a swelling would appear between the hip and costal margin and subside with the passage of a large amount of urine. In January, 1888, the sac discharged through the right lung.* Reaccumulations occurred on several occasions, and on June 9, 1891, the sac was opened and drained. He remains well, though there is still a sinus through which a clear, probably urinous, fluid is discharged.

The sac may discharge spontaneously through the ureter and the fluid never reaccumulate. In bilateral hydronephrosis there is a danger that uræmia may supervene. There are instances, too, in which blocking of the ureter on the sound side by calculus has been followed by uræmia. And, lastly, the sac may suppurate, and the condition change to one of pyonephrosis.

Treatment.—Cases of intermittent hydronephrosis which do not cause serious symptoms should be let alone. It is stated that, in sacs of moderate size, the obstruction has been overcome by shampooing. If practised, it should be done with great care. When the sac reaches a large size aspiration may be performed and repeated if necessary. Puncture should be made in the flank, midway between the ilium and the last rib. If the fluid reaccumulates and the sac becomes large, it may be incised and drained, or, as a last resort, the kidney may be removed. In women a carefully adapted pad and bandage will sometimes prevent the recurrence of an intermittent hydronephrosis.†

XI. NEPHROLITHIASIS (*Renal Calculus*).

Definition.—The formation in the kidney or in its pelvis of concretions, by the deposition of certain of the solid constituents of the urine.

Etiology and Pathology.—In the kidney substance itself the separation of the urinary salts produces a condition to which, unfortunately, the term infarct has been applied. Three varieties may be recognized: (1) The uric-acid infarct, usually met with at the apices of the pyramids in

* Sowers, New York Medical Record, 1888.

† See illustrative cases in my Lectures on Abdominal Tumors, 1894.

new-born children and during the first weeks of life. It is readily recognized as a yellowish linear streak in the pyramids and is of no significance; (2) the urate of soda infarct, sometimes associated with urate of ammonia, which forms whitish lines at the apices of the pyramids and is met with chiefly, but not always, in gouty persons; and (3) the lime infarcts, forming very opaque white lines in the pyramids, usually in old people.

In the pelvis and calyces concretions of the following forms occur: (*a*) Small gritty particles, *renal sand*, ranging in size from the individual grains of the uric-acid sediment to bodies 1 or 2 mm. in diameter. These may be passed in the urine for long periods without producing any symptoms, since they are too fine to be arrested in their downward passage.

(*b*) Larger concretions, ranging in size from a small pea to a bean, and either solitary or multiple in the calyces and pelvis. It is the smaller of these calculi which, in their passage, produce the attacks of renal colic. They may be rounded and smooth, or present numerous irregular projections.

(*c*) The dendritic form of calculus. The orifice of the ureter may be blocked by a Y-shaped stone. The pelvis itself may be occupied by the concretion, which forms a more or less distinct mould. These are the remarkable *coral calculi*, which form in the pelvis complete moulds of infundibula and calyces, the latter even presenting cup-like depressions corresponding to the apices of the papillæ. Some of these casts in stone of the renal pelvis are as beautifully moulded as Hyrtl's corrosion preparations.

Chemically the varieties of calculi are: (1) Uric acid, by far the most important, which may form the renal sand, the small solitary, or the large dendritic stones. They are very hard, the surface is smooth, and the color reddish. The larger stones are usually stratified and very dense. Usually the uric acid and the urates are mixed, but in children stones composed of urates alone may occur.

(2) Oxalate of lime, which forms mulberry-shaped calculi, studded with points and spines. They are often very dark in color, intensely hard, and are a mixture of oxalate of lime and uric acid.

(3) Phosphatic calculi are composed of the phosphate of lime and the ammonio-magnesium phosphate, sometimes mixed with a small amount of carbonate of lime. They are not common, since the phosphatic salts are oftener deposited about the uric acid or the oxalate of lime stones.

(4) Rare forms of calculi are made up of cystine, xanthine, carbonate of lime, indigo, and urostealith.

The mode of formation of calculi has been much discussed. They may be produced by an excess of a sparingly soluble abnormal ingredient, such as cystine or xanthine; more frequently by the presence of uric acid in a very acid urine which favors its deposition. Sir William Roberts thus briefly states the conditions which lead to the formation of the uric-acid concretions: high acidity, poverty in salines, low pigmentation, and high percentage of uric acid. The presence of albumin and mucus may determine, as Ord suggests, the deposition of the uric acid and thus form the starting point of a stone. Ova of parasites, blood-clots, casts, and shreds of

The madness and allied [illegible] ... have been attributed to various [illegible] (Smith Haworth)

epithelium may form the nuclei of stones. The question of bacterial infection has to be considered, as in the case of gall-stones.

Renal calculi are most common in the early and later periods of life. They are moderately frequent in this country, but there do not appear to be special districts, corresponding to the "stone counties" in England. Men are more often affected than women. Sedentary occupations seem to predispose to stone.

The effects of the calculi are varied. It is by no means uncommon to find a dozen or more stones of various sizes in the calyces without any destruction of the mucous membrane or dilatation of the pelvis. A turbid urine fills the pelvis in which there are numerous cells from the epithelial lining. There are cases of this sort in which, apparently, the stones may go on forming and are passed for years without seriously impairing the health and without inconvenience, except the attacks of renal colic. Still more remarkable are the cases of coral-like calculi, which may occupy the entire pelvis and calyces without causing pyelitis, but which gradually lead to more or less induration of the kidney. The most serious effects are when the stone excites a suppurative pyelitis and pyonephrosis.

Symptoms.—Patients may pass gravel for years without having an attack of renal colic, and a stone may never lodge in the ureter. In other instances, the formation of calculi goes on year by year and the patient has recurring attacks such as have been so graphically described by Montaigne. in his own case. A patient may pass an enormous number of calculi. Some years ago I was consulted by a commercial traveller, an extremely vigorous man, who for many years had had repeated attacks of renal colic, and had passed several hundred calculi of various sizes. His collection filled an ounce bottle. A patient may pass a single calculus, and never be troubled again. The large coral calculi may excite no symptoms. In a remarkable specimen of the kind, presented to the McGill Medical Museum by J. A. Macdonald, the patient, a middle-aged woman, died suddenly with uræmic symptoms. There was no pyelitis, but the kidneys were sclerotic.

Renal colic ensues when a stone enters the ureter. An attack may set in abruptly without apparent cause, or may follow a strain in lifting. It is characterized by agonizing pain, which starts in the flank of the affected side, passes down the ureter, and is felt in the testicle and along the inner side of the thigh. The pain may also radiate through the abdomen and chest, and be very intense in the back. In severe attacks there are nausea and vomiting and the patient is collapsed. The perspiration breaks out upon the face and the pulse is feeble and quick. A chill may precede the outbreak, and the temperature may rise as high as 103°. No one has more graphically described an attack of "the stone" than Montaigne,* who was a sufferer for many years: "Thou art seen to sweat with pain, to look pale and red, to tremble, to vomit well-nigh to blood, to suffer strange contortions and convulsions, by starts to let tears drop from thine eyes, to urine thick, black, and frightful water, or to have it suppressed by some sharp and craggy stone, that cruelly pricks and tears thee." The

* Essays, Book III, 13.

symptoms persist for a variable period. In short attacks they do not last longer than an hour; in other instances they continue for a day or more, with temporary relief. Micturition is frequent, occasionally painful, and the urine, as a rule, is bloody. There are instances in which a large amount of clear urine is passed, probably from the other kidney. In rare cases the secretion of urine is completely suppressed, even when the kidney on the opposite side is normal, and death may occur from uræmia. This most frequently happens when the second kidney is extensively diseased, or when only a single kidney exists. A number of cases of this kind have been recorded. The condition has been termed, by Sir William Roberts, obstructive suppression. It is met with also when cancer compresses both ureters or involves their orifices in the bladder. The patient may not appear to be seriously ill at first, and uræmic symptoms may not develop for a week, when twitching of the muscles, great restlessness, and sometimes drowsiness supervene, but, strange to say, neither convulsions nor coma. Death takes place usually within twelve days from the onset of the obstruction.

After the attack of colic has passed there is more or less aching on the affected side, and the patient can usually tell from which kidney the stone has come. Examination during the attack is usually negative. Very rarely the kidney becomes palpable. Tenderness on the affected side is common. In very thin persons it may be possible, on examination of the abdomen, to feel the stone in the ureter; or the patient may complain of a grating sensation.

When the calculi remain in the kidney they may produce very definite and characteristic symptoms, of which the following are the most important:

(1) *Pain*, usually in the back, which is often no more than a dull soreness, but which may be severe and come on in paroxysms. It is usually on the side affected, but may be referred to the opposite kidney, and there are instances in which the pain has been confined to the sound side. Pains of a similar nature may occur in movable kidneys, and there are several instances on record in which surgeons have incised the kidney for stone and found none. In an instance in which pain was present for a couple of years the exploration revealed only a contracted kidney.

(2) *Hæmaturia.*—Although this occurs most frequently when the stone becomes engaged in the ureter, it may also come on when the stones are in the pelvis. The bleeding is seldom profuse, as in cancer, but in some instances may persist for a long time. It is aggravated by exertion and lessened by rest. Frequently it only gives to the urine a smoky hue. The urine may be free for days, and then a sudden exertion or a prolonged ride may cause smokiness, or blood may be passed in considerable quantities.

(3) *Pyelitis.*—(*a*) There may be attacks of severe pain in the back, not amounting to actual colic, which are initiated by a heavy chill followed by fever, in which the temperature may reach 104° or 105°, followed by profuse sweating. The urine, which has been clear, may become turbid and smoky and contain blood and abundant epithelium from the pelvis. Attacks of this description may recur at intervals for months or even

years, and are generally mistaken for malaria, unless special attention is paid to the urine and to the existence of the pain in the back. This renal intermittent fever, due to the presence of calculi, is analogous to the hepatic intermittent fever, due to gall-stones, and in both it is important to remember that the most intense paroxysms may occur without any evidence of suppuration.

(*b*) More frequently the symptoms of purulent pyelitis, which have already been described, are present; pain in the renal region, recurring chills, and pus in the urine, with or without indications of pyonephrosis.

(4) *Pyuria.*—There are instances of stone in the kidney in which pus occurs continuously or intermittently in the urine for many years. On many occasions between 1875 and 1884 I examined the urine of a physician who had passed calculi when a student in 1845, and has had pus in the urine at intervals to 1891. In spite of the prolonged suppuration he has had remarkable mental and bodily vigor.

Patients with stone in the kidney are often robust, high livers, and gouty. Attacks of dyspepsia are not uncommon, or they may have severe headaches.

Diagnosis.—Renal may be mistaken for intestinal colic, particularly if the distention of the bowels is marked, or for biliary colic. The situation and direction of the pain, the retraction and tenderness of the testicle, the occurrence of hæmaturia, and the altered character of the urine are distinctive features. Attention may again be called to the fact that attacks simulating renal colic are associated with movable kidney, or even, it has been supposed, without mobility of the kidney, with the accumulation of the oxalates or uric acid in the pelvis of the kidney. The diagnosis between a stone in the kidney and stone in the bladder is not always easy, though in the latter the pain is particularly about the neck of the bladder, and not limited to one side. Important points are the reaction of the urine, which in stone in the bladder is almost invariably alkaline, and the abundance of mucus with the pus. It is stated that certain differences occur in the symptoms produced by different sorts of calculi. The large uric-acid calculi less frequently produce severe symptoms. On the other hand, as the oxalate of lime is a rougher calculus, it is apt to produce more pain (often of a radiating character) than the lithic-acid form, and to cause hæmorrhage. In both these forms the urine is acid. The phosphatic calculi are stated to produce the most intense pain, and the urine is commonly alkaline. In a few cases the Roentgen rays have been of use in determining the presence of a stone.

Treatment.—In the attacks of renal colic great relief is experienced by the hot bath, which is sometimes sufficient to relax the spasm. When the pain is very intense morphia should be given hypodermically, and inhalations of chloroform may be necessary until the effects of the anodyne are manifest. Local applications are sometimes grateful—hot poultices, or cloths wrung out of hot water. The patient may drink freely of hot lemonade, soda water, or barley water. Occasionally change in posture will give great relief, and inversion of the patient is said to be followed by immediate cessation of the pain.

In the intervals the patient should, as far as possible, live a quiet life, avoiding sudden exertion of all sorts. The essential feature in the treatment is to keep the urine abundant and, in a majority of the cases, alkaline. The patient should drink daily a large but definite quantity of mineral waters * or distilled water, which is just as satisfactory. The citrate or bicarbonate of potash may be added. The aching pains in the back are often greatly relieved by this treatment. Many patients find benefit from a stay at Saratoga, Bedford, Poland, or other mineral springs in this country, or at Vichy or Ems in Europe.

The diet should be carefully regulated, and similar to that indicated in the early stages of gout. Sir William Roberts recommends what is known as the solvent treatment for uric-acid calculi. The citrate of potash is given in large doses, half a drachm to a drachm, every three hours in a tumblerful of water. This should be kept up for several months. I have had no success with this treatment, nor, when one considers the character of the uric-acid stones usually met with in the kidney, does it seem likely that any solvent action could be exercised upon them by changes in the urine. This treatment should be abandoned if the urine becomes ammoniacal.

The value of piperazine as a solvent of uric-acid gravel or of uric-acid stones has been much discussed of late. While outside the body a watery solution of the drug has this power in a marked degree, the amount excreted in the urine as given in the ordinary doses of 15 grains daily seems to have very little influence. Several observers have shown that the percentage of piperazine excreted in the urine, when taken in doses of from 1 to 2 grammes, has, when tested outside of the body, little or no influence as a solvent (Fawcett, Gordon).

XII. TUMORS OF THE KIDNEY.

These are benign and malignant. Of the benign tumors, the most common are the small nodular *fibromata* which occur frequently in the pyramids, the *aberrant adrenals*, which Grawitz has described, and occasionally *lipoma*, *angioma*, or *lymphadenoma*. The *adenomata* may be congenital. In one of my cases the kidneys were greatly enlarged, contained small cysts, and numerous adenomatous structures throughout both organs.

Malignant growths—*cancer* or *sarcoma*—may be either primary or secondary. The sarcomata are the most common, either alveolar sarcoma or the remarkable form containing striped muscular fibres—rhabdo-myoma. They are very common tumors in children. G. Walker (Annals of Surgery, 1897) has analyzed the literature of the subject to date. Carcinoma is less frequent, and is of the encephaloid variety.

The tumors attain a very large size. In one of my cases the left kidney weighed 12 pounds and almost filled the abdomen. In children they may

* Some of these, if we judge by the laudatory reports, are as potent as the waters of Corsena, declared by Montaigne to be "powerful enough to break stones."

reach an enormous size. Morris states that in a boy at the Middlesex Hospital the tumor weighed 31 pounds. They grow rapidly, are often soft, and hæmorrhage frequently takes place into them. In the sarcomata, invasion of the pelvis or of the renal vein is common. The rhabdo-myomas rarely form very large tumors, and death occurs shortly after birth. In one of my cases the child lived to the age of three years and a half. The tumor grew into the renal vein and inferior cava. A detached fragment passed as an embolus into the pulmonary artery, and a portion of it blocked the tricuspid orifice.

Symptoms.—The following are the most important: (1) Hæmaturia. This may be the first indication. The blood is fluid or clotted, and there may be very characteristic moulds of the pelvis of the kidney and of the ureter. It would no doubt be possible for such to form in the hæmaturia from calculus, but I have never met with a case of blood-casts of the pelvis and of the ureter, either alone or together, except in cancer. It is rare indeed that cancer elements can be recognized in the urine.

(2) Pain is an uncertain symptom. In several of the largest tumors which have come under my observation there has been no discomfort from beginning to close. When present, it is of a dragging, dull character, situated in the flank and radiating down the thigh. The passage of the clots may cause great pain. In a recent case the growth was at first upward, and the symptoms for some months were those of pleurisy.

(3) Progressive emaciation. The loss of flesh is usually marked and advances rapidly. There may, however, be a very large tumor without emaciation.

Physical Signs.—In almost all instances tumor is present. When small and on the right side, it may be very movable; in some instances, occupying a position in the iliac fossa, it has been mistaken for ovarian tumor. The large growths fill the flank and gradually extend toward the middle line, occupying the right or left half of the abdomen. Inspection may show two or three hemispherical projections corresponding to distended sections of the organ. In children the abdomen may reach an enormous size and the veins are prominent and distended. On bimanual palpation the tumor is felt to occupy the lumbar region and can usually be lifted slightly from its bed; in some cases it is very movable, even when large; in others it is fixed, firm, and solid. The respiratory movements have but slight influence upon it. Rapidly growing renal tumors are soft, and on palpation may give a sense of fluctuation. A point of considerable importance is the fact that the colon crosses the tumor, and can usually be detected without difficulty.

Diagnosis.—In children very large abdominal tumors are either renal or retroperitoneal. The retroperitoneal sarcoma (Lobstein's cancer) is more central, but may attain as large a size. If the case is seen only toward the end, a differential diagnosis may be impossible; but as a rule the sarcoma is less movable. It is to be remembered that these tumors may invade the kidney. On the left side an enlarged spleen is readily distinguished, as the edge is very distinct and the notch or notches well marked; it descends during respiration, and the colon lies behind, not in front of it. On the

right side growths of the liver are occasionally confounded with renal tumors; but such instances are rare, and there can usually be detected a zone of resonance between the upper margin of the renal tumor and the ribs. Late in the disease, however, this is not possible, for the renal tumor is in close union with the liver.

A malignant growth in a movable kidney may be very deceptive and may simulate cancer of the ovary or myoma of the uterus. The great mobility upward of the renal growth and the negative result of examination of the pelvic viscera are the reliable points.

Medicinal treatment is of no avail. When the growth is small and the patient in good condition removal of the organ may be undertaken, but the percentage of cases of recovery is very small, only 5.4 per cent (G. Walker).

XIII. CYSTIC DISEASE OF THE KIDNEY.

The following varieties of cysts are met with:

(1) The small cysts, already described in connection with the chronic nephritis, which result from dilatation of obstructed tubules or of Bowman's capsules. There are cases very difficult to classify, in which the kidneys are greatly enlarged, and very cystic in middle-aged or elderly persons, and yet not so large as in the congenital form.

(2) Solitary cysts, ranging in size from a marble to an orange, or even larger, are occasionally found in kidneys which present no other changes. In exceptional cases, they may form tumors of considerable size. Newman operated on one which contained 25 ounces of blood. They, too, in all probability, result from obstruction.

(3) The congenital cystic kidneys. In this remarkable condition the kidneys are represented by a conglomeration of cysts, varying in size from a pea to a marble. The organs are greatly enlarged, and together may weigh 6 or more pounds. In the fœtus they may attain a size sufficient to impede labor. Little or no renal tissue may be noticeable, although in microscopical sections it is seen that a considerable amount remains in the interspaces. The cysts contain a clear or turbid fluid, sometimes reddish brown or even blackish in color, and may be of a colloidal consistence. Albumin, blood crystals, cholesterin, with triple phosphates and fat drops are found in the contents. Urea and uric acid are rarely present. The cysts are lined by a flattened epithelium. It is not yet accurately known how these cysts originate. That it is a defect in development rather than a pathological change is suggested by the fact that in the embryo it is often associated with other anomalies, particularly imperforate anus. Both Shattock and Bland Sutton, who have studied the question carefully, believe that the anomaly of development is in the failure of complete differentiation of the Wolffian bodies, which are, as it were, mixed with the kidneys and give rise to the cysts. Though the condition is congenital, yet from the history of certain cases it is evident that the organs must increase enormously in size. In a patient of Dr. Alfred King's, of Portland, Me., a man aged fifty-four, the abdomen presented nothing abnormal on careful

examination three years before his death, but three months prior to this date there were large bilateral tumors in the renal regions, which were readily diagnosed as cystic kidneys. The organs weighed 4 pounds each.

In a large majority of the cases death occurs, either *in utero* or shortly after birth; but instances are met with at all ages up to fifty or sixty, and I see no reason to suppose that these are not instances of persistence of the congenital form.

In the adult the tumors may be felt in the lumbar region as large rounded masses.

The *symptoms* are those of chronic interstitial nephritis. Many of the cases have presented no indications whatever until a sudden attack of uræmia; others have died of heart-failure. A rare termination, as in a case at the University Hospital, Philadelphia, is the rupture of one of the cysts and the production of a perinephritic abscess. The cardio-vascular changes induced are similar to those of interstitial nephritis. The left ventricle is hypertrophied and the arterial tension is greatly increased. The condition is compatible with excellent health. Hæmaturia may occur. The dangers are those associated with chronic Bright's disease. It is important to remember that the conglomerate cystic kidney is almost invariably bilateral. One kidney may be somewhat larger and more cystic than the other.

The diagnosis can sometimes be made. Great enlargement of both organs, with hypertrophy of the left heart and increased arterial tension, would suggest the condition.

Operative interference is not justifiable. I know of an instance in which one kidney was removed and the patient died within twenty-four hours.

(4) Occasionally the kidneys and liver present numerous small cysts scattered through the substance. The spleen and the thyroid also may be involved, and there may be congenital malformation of the heart. The cysts in the kidney are small, and neither so numerous nor so thickly set as in the conglomerate form, though in these cases the condition is probably the result of some congenital defect. There are cases, however, in which the kidneys are very large. It is more common in the lower animals than in man. I have seen several instances of it in the hog; in one case the liver weighed 40 pounds, and was converted into a mass of simple cysts. The kidneys were less involved. Charles Kennedy * states that he has found references to 12 cases of combined cystic disease of the liver and kidneys.

The echinococcus cysts have been described under the section on parasites. Paranephric cysts (external to the capsule) are rare; they may reach a large size.

* Laboratory Reports of the Royal College of Physicians, Edinburgh, vol. iii.

XIV. PERINEPHRIC ABSCESS.

Suppuration in the connective tissue about the kidney may follow (1) blows and injuries; (2) the extension of inflammation from the pelvis of the kidney, the kidney itself, or the ureters; (3) perforation of the bowel, most commonly the appendix, in some instances the colon; (4) extension of suppuration from the spine, as in caries, or from the pleura, as in empyema; (5) as a sequel of the fevers, particularly in children.

Post mortem the kidney is surrounded by pus, particularly at the posterior part, though the pus may lie altogether in front, between the kidney and the peritonæum. Usually the abscess cavity is extensive. The pus is often offensive and may have a distinctly fæcal odor from contact with the large bowel. It may burrow in various directions and burst into the pleura and be discharged through the lungs. A more frequent direction is down the psoas muscle, when it appears in the groin, or it may pass along the iliacus fascia and appear at Poupart's ligament. It may perforate the bowel or rupture into the peritonæum; sometimes it penetrates the bladder or vagina.

Post mortem we occasionally find a condition of chronic perinephritis in which the fatty capsule of the kidney is extremely firm, with numerous bands of fibrous tissue, and is stripped off from the proper capsule with the greatest difficulty. Such a condition probably produces no symptoms.

Symptoms.—There may be intense pain, aggravated by pressure, in the lumbar region. In other instances, the onset is insidious, without pain in the renal region; on examination signs of deep-seated suppuration may be detected. On the affected side there is usually pain, which may be referred to the neighborhood of the hip-joint or to the joint itself, or radiate down the thigh and be associated with retraction of the testis. The patient lies with the thigh flexed, so as to relax the psoas muscle, and in walking throws, as far as possible, the weight on the opposite leg. The patient keeps the spine immobile, assumes a stooping posture in walking, and has great difficulty in voluntarily adducting the thigh (Gibney).

There may be pus in the urine if the disease has extended from the pelvis or the kidney, but in other forms the urine is clear. When pus has formed there are usually chills with irregular fever and sweats. On examination, deep-seated induration is felt between the last rib and the crest of the ilium. Bimanual palpation may reveal a distinct tumor mass. Œdema or puffiness of the skin is frequently present.

The *diagnosis* is usually easy; when doubt exists the aspirator needle should be used. We cannot always differentiate the primary forms from those due to perforation of the kidney or of the bowel. This, however, makes but little difference, for the treatment is identical. It is usually possible by the history and examination to exclude diseases of the vertebra. In children hip-joint disease may be suspected, but the pain is higher, and there is no fulness or tenderness over the hip-joint itself.

The *treatment* is clear—early, free, and permanent drainage.

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

I. GENERAL INTRODUCTION.

In diseases of the nervous system it is of the greatest importance to know accurately the position of the morbid process, and here, even more than in the other departments of medicine, a thorough knowledge of anatomy and physiology is essential. For full details the student is referred to the text-books on the subject, as it is not possible to do more than touch on the subject in this place.

Recent studies have modified our conceptions of the fundamental structure of the nervous system. At present we think of it as a combination of an immense number of units, called *neurones*, all having an essentially similar structure. Each neurone is composed of a cell body, the protoplasmic processes or dendrites, and the axis-cylinder process or axone. The nutrition of the neurone depends in large part upon the condition of the cell body, and this in turn in all probability upon the activity of the nucleus. If the cell is injured in any manner the processes degenerate, or if the processes are separated from the cell they degenerate. Whether or not the neurones are organically connected with one another is still in dispute. The weight of evidence is in favor of complete anatomical and relative physiological independence. The terminals of the axone of one neurone are related to the dendrites and cell bodies of other neurones by contact (Ramón y Cajal) or by concrescence (Held). It is generally admitted, however, that occasional coarse anastomoses exist between neighboring dendrites (according to Dogiel), especially in the retina. The studies of Apáthy speak in favor of a general interconnection by means of neurofibrils and protoplasmic bridges. In general, it may be stated that the dendrites or protoplasmic processes conduct impulses toward the cell body (cellulipetal conduction), and the axis-cylinder process conducts them away from the cell (cellulifugal conduction). The axis-cylinder process after leaving the cell gives off at varying intervals lateral branches called collaterals, which run at right angles to the process. The collaterals and finally the axis-cylinder process itself at their terminations split up into many fine fibres, forming the end-brushes. These, known as arborizations, surround the body of one or more

of the many other cells, or interlace with their protoplasmic processes. The cell bodies of the neurones are collected more or less closely together in the gray matter of the brain and spinal cord and in the ganglia of the peripheral nerves. Their processes, especially the axis-cylinder processes, run for the most part in the white tracts of the brain and spinal cord and in the peripheral nerves. In this way the different parts of the central nervous system are brought into relation with each other and with the rest of the body. In many cases the connections are extremely complicated and have only just begun to be unravelled, but, fortunately for the clinician, the nervous mechanism upon which motion depends is the best understood and is the simplest.

A voluntary motor impulse starting from the brain cortex must pass through at least two neurones before it can reach the muscles, and we therefore speak of the motor tract as being composed of two segments—an upper and a lower. The neurones of the lower segment have the cell bodies and their protoplasmic processes in the different levels of the ventral horns of the spinal cord and in the motor nuclei of the cerebral nerves. The axis-cylinder processes of the lower motor neurones leave the spinal cord in the ventral roots and run in the peripheral nerves, to be distributed to all the muscles of the body, where they end in arborizations in the motor end plates. These neurones are direct—that is, their cell bodies, their processes, and the muscles in which they end are all on the same side of the body.*

The neurones of the upper motor segment have their cell bodies and protoplasmic processes in the cortex of the brain about the fissure of Rolando. Their axis-cylinder processes run in the white matter of the brain through the internal capsule and the cerebral peduncles into the pons, medulla, and cord, ending in arborizations around the protoplasmic processes and cell bodies of the lower motor neurones. The upper segment is, in the main, a crossed tract—that is to say, the neurones which compose it have their protoplasmic processes and cell bodies on one side of the body, whereas their axis-cylinder processes cross the middle line, to end about cell bodies of the lower motor neurones on the opposite side of the body. A certain number of the axones of the pyramidal tract, however, run to the lower motor neurones of the same side.

Motor impulses starting in the left side of the brain cause contractions of muscles on the right side of the body, and those from the right side of the brain in muscles of the left side of the body. Leaving out of consideration the exceptions which have been mentioned, it may be stated as a general rule that the motor path is crossed, and that the crossing takes place in the upper segment (Figs. 1 and 2). Every muscular movement, even the simplest, requires the activity of many neurones. In the production of each movement special neurones are brought into play in a definite combination, and whenever these neurones act in this combination that specific movement is the result. In other words, all the movements of the

* The root fibres of the nervus trochlearis and a portion of the root fibres of the nervus oculomotorius are well-known exceptions to this rule.

body are represented in the central nervous system by combinations of neurones—that is, they are localized. Muscular movements are localized in every part of the motor path, and in cases of disease of the nervous system a study of the motor defect often enables one to fix upon the site of the process, and it would be hard to overestimate the importance of a thorough knowledge of such localization.

The axis-cylinder processes of the lower motor neurones run in the peripheral nerves. Each nerve contains processes which are supplied to definite muscles, and we have in this way a peripheral localization. (See sections on Diseases of the Cerebral and Spinal Nerves.)

The axis-cylinder processes which run in the peripheral nerves leave the central nervous system from its ventral aspect. The ventral roots of the spinal cord are from above down, collected into small groups, which, after joining with the dorsal roots of the same level of the cord, leave the spinal canal between the vertebræ as the spinal nerves. That part of the cord from which the roots forming a single spinal nerve arise is called a segment, and corresponds to the nerve which arises from it and not to the vertebra to which it may be opposite. The axis-cylinder processes which go to make up any one peripheral nerve do not necessarily arise from the same segment of the spinal cord; in fact, most peripheral nerves contain processes from several often quite widely separated segments, and so it happens that the movements are represented in the spinal cord in a different manner—that is, there is spinal localization, or, better, lower level localization, since it also includes the motor nuclei of the cerebral nerves.

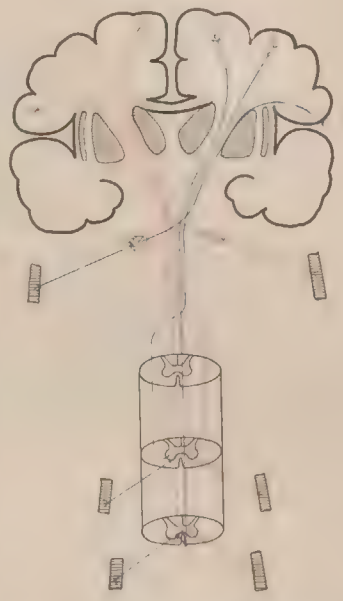

Fig. 1.—Diagram of motor path, showing the crossing of the path, which takes place in the upper segment. (Van Gehuchten, colored.)

Our knowledge of the localization of the muscular movements in the gray matter of the lower motor segment is far from complete, but enough is known to aid materially in determining the site of a spinal lesion. A number of tables have been prepared by different observers to represent our present knowledge of this subject. They differ from each other in minor details, but agree in the main. The following is the table prepared by Starr, in which the names of the muscles are given whose movements are represented in each of the spinal segments. Movements, not muscles, are localized in the central nervous system, a point carefully to be borne in mind by the student.

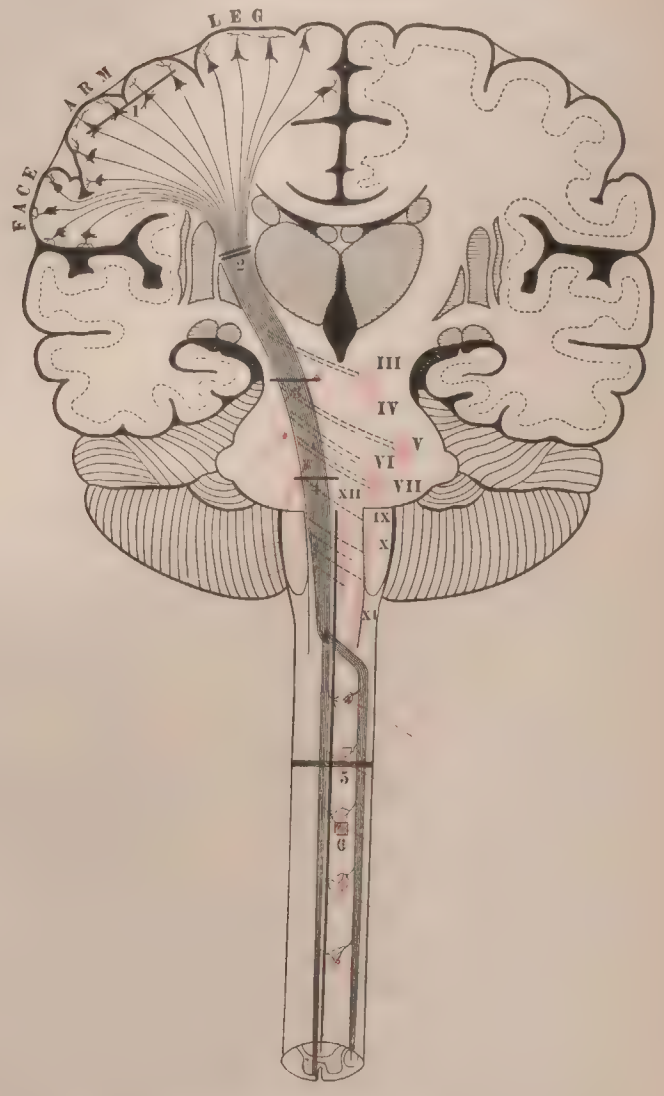

FIG. 2.—Diagram of motor path from right brain. The upper segment is black, the lower red. The nuclei of the motor cerebral nerves are shown on the left side: on the right side the cerebral nerves of that side are indicated. A lesion at 1 would cause upper segment paralysis in the arm of the opposite side—cerebral monoplegia; at 2, upper segment paralysis of the whole opposite side of the body—hemiplegia; at 3, upper segment paralysis of the opposite face, arm, and leg, and lower segment paralysis of the eye muscles on the same side—crossed paralysis; at 4, upper segment paralysis of opposite arm and leg, and lower segment paralysis of the face and the external rectus on the same side—crossed paralysis; at 5, upper segment paralysis of all muscles below lesion, and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6, lower segment paralysis of muscles localized at seat of lesion—anterior poliomyelitis. (Van Gehuchten, modified.)

Localization of the Functions of the Segments of the Spinal Cord.

Segment.	Muscles.	Reflex.	Sensation.
II and III C.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to 1st thoracic. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front and back.
VI C.	Biceps. Brachialis anticus. Pectoralis (clavicular part). Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front and back. Outer half of hand.
VII C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar. 7th cervical to 1st thoracic. Stroking palm causes closure of fingers.	Inner side and back of arm and forearm. Radial half of the hand.
VIII C.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I T.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II to XII T.	Muscles of back and abdomen. Erectores spinæ.	Epigastric. 4th to 7th thoracic. Tickling mammary regions causes retraction of epigastrium. Abdominal. 7th to 11th thoracic. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downward, corresponding to spinal nerves. Upper gluteal region.

Segment.	Muscles.	Reflex.	Sensation.
I L.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
II L.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patellar tendon. Tapping tendon causes extension of leg.	Outer side of thigh.
III L.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Front and inner side of thigh.
IV L.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle. Inner side of foot.
V L.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot.
I to II S.	Flexors of ankle. Long flexor of toes. Peronæi. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh. Leg and foot, outer side.
III to V S.	Perineal muscles.	Foot reflex. Achilles tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Skin over sacrum. Anus. Perinæum. Genitals.

The above table refers only to localization in the spinal cord. The manner in which movements are represented in the pons and medulla is about as follows. This table is constructed from above downward in reference to the motor nuclei of the cranial nerves:

Nuclei.

III. Sphincter. Ciliary muscles. Levator palpebræ superioris. Rectus internus (in convergence). Rectus superior. Rectus inferior.

IV. Obliquus inferior. Obliquus superior.

(Upper facial group.)

V. (Associated movement of levator palpebræ.) Muscles of lower jaw.

VI. Rectus externus. Rectus inter. of opposite side in lateral movements.

VII.—Facial muscles.

XII. (Lower facial group.) Muscles of tongue.

IX. Muscles of pharynx.

X. Muscles of œsophagus.

XI. Muscles of larynx.

Cerebral Motor Localization.—The cell bodies of the upper motor neurones are found in the brain cortex about the fissure of Rolando, and it is in this region that we find the movements of the body again represented.

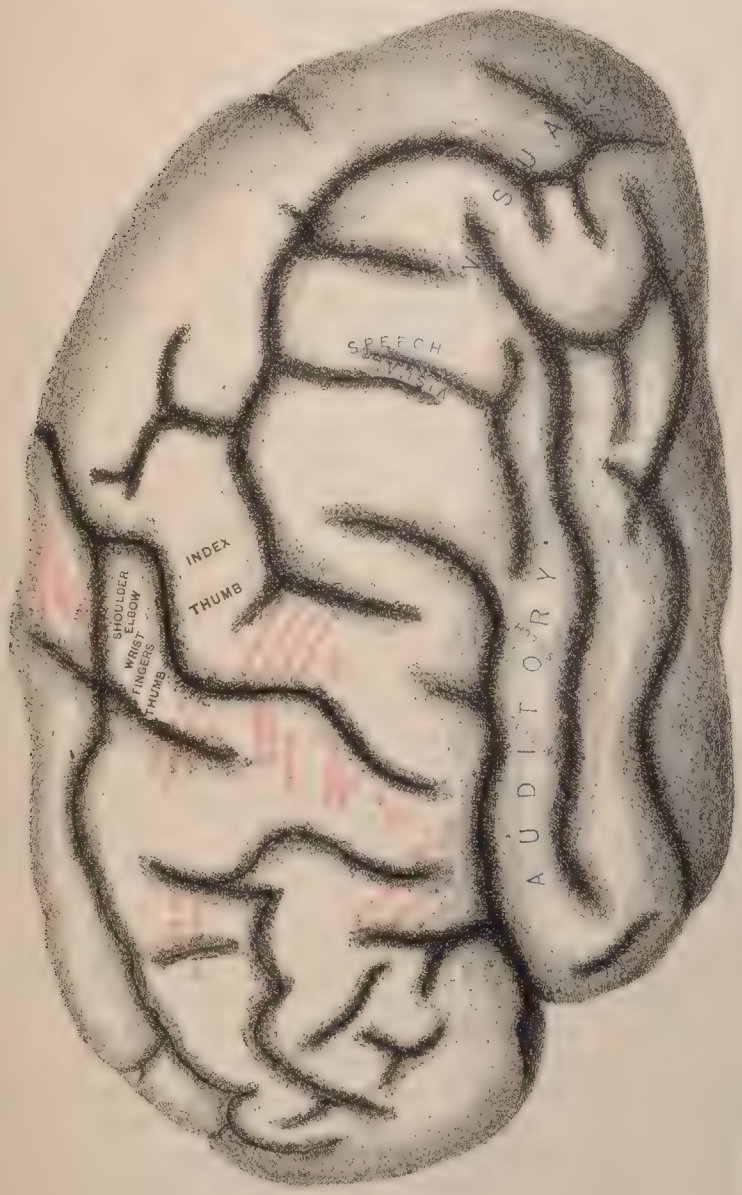

FIG. 3.—Diagram of cerebral localization. The motor areas determined by Beevor and Horsley on the orang-outang have been transferred approximately to Schäfer's drawing (Quain's Anatomy) somewhat simplified. The areas of the representation of the cutaneous sensations are too little known to be included, and those of auditory and visual sensations are merely indicated, but their boundaries are not accurately defined.

The clinical studies of Hughlings Jackson, and the experiments of Hitzig and Fritsch, and of Ferrier, laid the foundation for the great mass of most excellent work which has been done upon this subject. We owe much

to Victor Horsley and his associates for their careful work in this direction, and the following description is based largely upon their writings, and especially upon the paper of Beevor and Horsley, in which they give the results of their experimental work on the orang-outang. Clinical observation and electrical stimulation of the brain cortex during operations on human beings have confirmed the results of experiments upon animals.

The motor area comprises the anterior central convolution, and to a less extent the posterior central convolution, the hinder part of the three frontal convolutions and the paracentral lobule. In the orang-outang and man not every part of this region is excitable by electrical stimulation. The movements are quite sharply localized, and there are inexcitable areas between the areas of representation of the larger divisions of the body. The diagram (Fig. 3) shows the centres as given by Beevor and Horsley. Certain landmarks are important. The genu of the fissure of Rolando, which when present in man is found at a point about midway or even higher between the upper margin of the hemisphere and the fissure of Sylvius, marks the boundary between the area of representation of the arm from that of the face. The level of the superior frontal sulcus indicates the division of the leg from the arm area. From above down the areas of representation occur in this order: leg, arm, face. Those of the leg and arm occupy the upper half of the convolution, and that for the face is spread out over the lower half. The diagram indicates the localization of the movements of the different parts of the extremities.

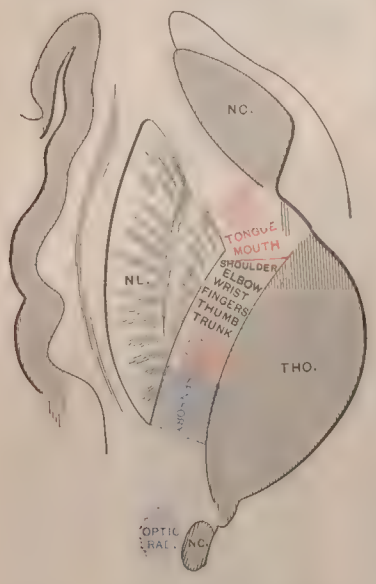

FIG. 4.—Diagram of motor and sensory representation in the internal capsule. NL., Lenticular nucleus. NC., Caudate nucleus. THO., Optic thalamus. The motor paths are red and black, the sensory are blue.

The centres for the trunk are, according to Schäfer, situated in the marginal gyrus just within the longitudinal fissure in the paracentral lobule. In man the motor speech centre is localized in the posterior part of the left inferior frontal convolution.

The axis-cylinder processes of the upper motor neurones after leaving the gray matter of the motor cortex pass into the white matter of the brain and form part of the corona radiata. They converge and pass between the basal ganglia in the internal capsule. Here the motor axis-cylinders are collected into a compact bundle—the pyramidal tract—occupying the knee and anterior two thirds of the posterior limb of the internal capsule. The order in which the movements of the opposite side of the body are represented here is given in Fig. 4.

After passing through the internal capsule the fibres of the pyramidal

tract leave the hemisphere by the crus, in which they occupy about the middle three fifths (Fig. 5). The movements of the tongue and lips are represented nearest the middle line.

As soon as the tract enters the crus, some of its axis-cylinder processes leave it and cross the middle line to end in arborizations about the ganglion cells in the nucleus of the third nerve on the opposite side; and in this way, as the pyramidal tract passes down, it gives off at different levels fibres which end in the nuclei of all the motor cerebral nerves on the opposite side of the body. Some fibres, however, go to the nuclei of the same side (Hoche). From the crus, the pyramidal tract runs through the pons and forms in the medulla oblongata the pyramid, which gives its name to the tract. At the lower part of the medulla, after the fibres going to the cerebral nerves have crossed the middle line, a large proportion of the remaining fibres cross, decussating with those from the opposite pyramid, and pass into the opposite side of the spinal cord, forming the crossed pyramidal tract of the lateral column (fasciculus cerebrospinalis lateralis) (Fig. 6, 1). The smaller number of fibres which do not at this time cross, descend in the ventral column of the same side, forming the direct pyramidal tract, or Türck's column (fasciculus cerebrospinalis ventralis) (Fig. 6, 2).

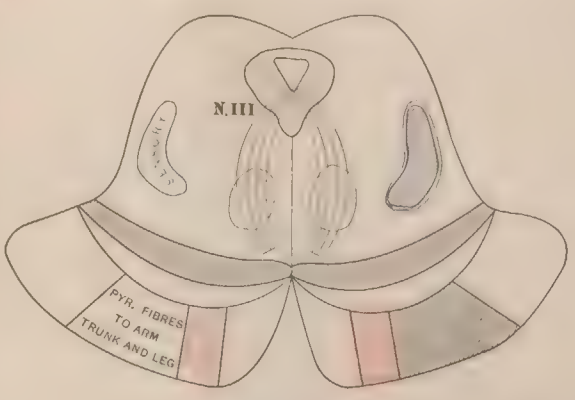

Fig. 5.—Diagram of motor and sensory paths in Crura.

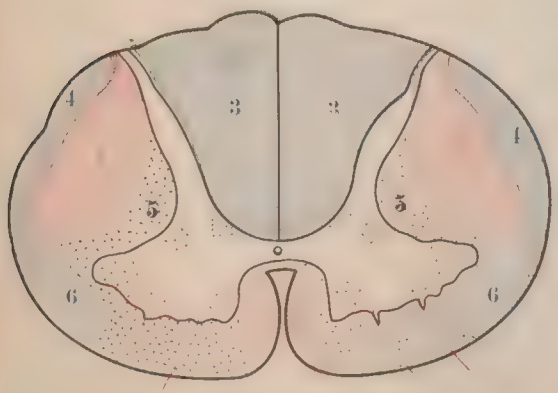

Fig. 6.—Diagram of cross-section of spinal cord, showing motor, red, and sensory, blue paths. 1, Lateral pyramidal tract. 2, Ventral pyramidal tract. 3, Dorsal columns. 4, Direct cerebellar tract. 5, Ventro-lateral ground bundles. 6, Ventro-lateral ascending tract of Gowers. (Van Gehuchten, colored.)

At every level of the spinal cord axis-cylinder processes leave the crossed pyramidal tract to enter the ventral horns and end about the cell bodies of the lower motor neurones. The tract diminishes in size from above downward. The fibres of the direct

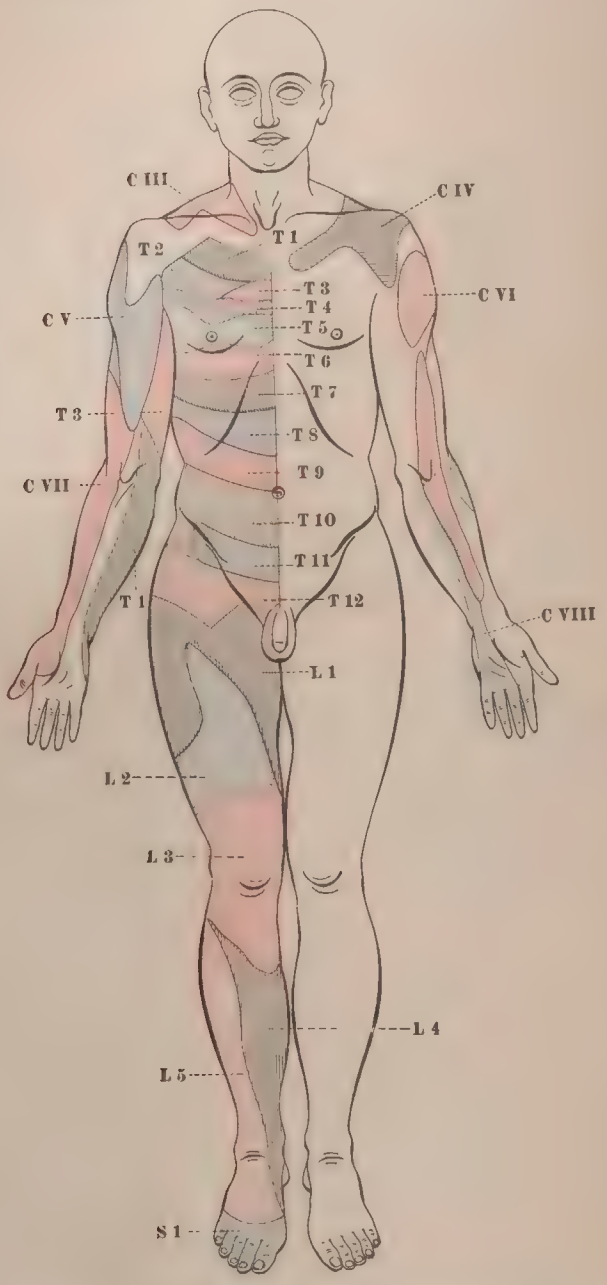

FIG. 7.—Diagram of skin areas corresponding to the different spinal segments. (Combined from Head's diagrams.)

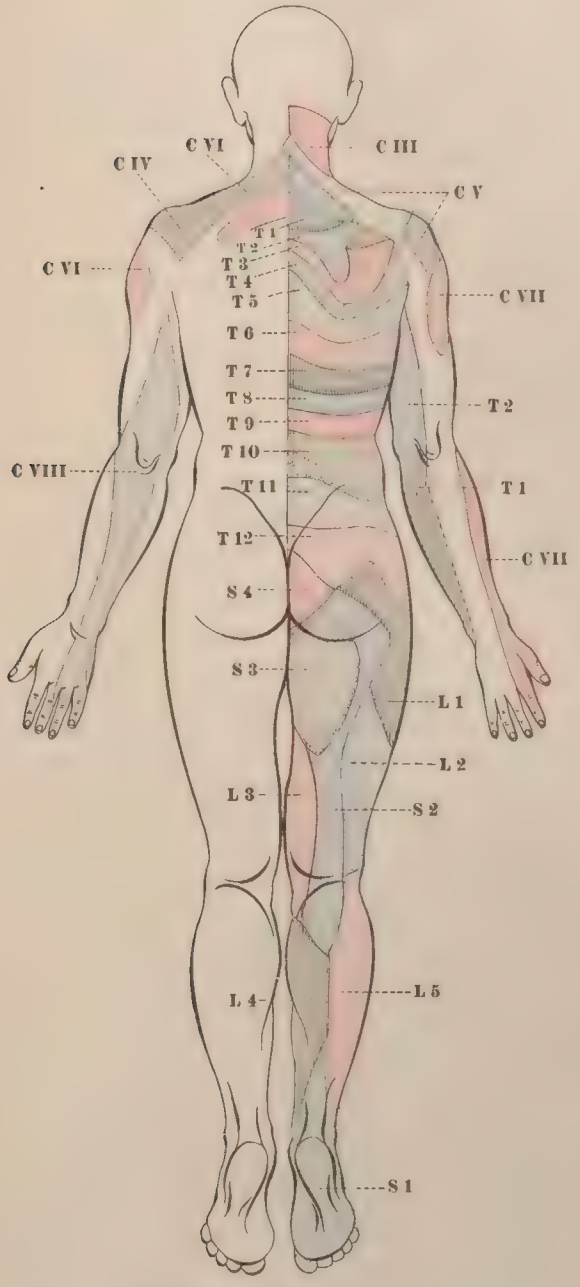

FIG. 8.—Diagram of skin areas corresponding to the different spinal segments. (Combined from Head's diagrams.)

pyramidal tract cross at different levels in the ventral white commissure, and also, it is believed, end about cells in the ventral horns on the opposite side of the cord. This tract usually ends about the middle of the thoracic region of the cord.

The path for sensory conduction is more complicated than the motor path, and in its simplest form is composed of at least three sets of neurones, one above the other. The cell bodies of the lowest neurones are in the ganglia, on the dorsal roots of the spinal nerves, and the ganglia of the sensory cerebral nerves. These ganglion cells have a special form, having apparently but a single process, which, soon after leaving the cell, divides in a T-shaped manner, one portion running into the central nervous system and the other to the periphery of the body. Embryological and comparative anatomical studies have made it probable that the peripheral sensory fibre, the process which conducts toward the cell, represents the protoplasmic processes, while that which conducts away from the cell is the axis-cylinder process. In the peripheral sensory nerves we have, then, the dendrites of the lower sensory neurones. These start in the periphery of the body from their various specialized end organs. The axis-cylinder processes leave the ganglia and enter the spinal cord by the dorsal roots of the spinal nerves. After entering the cord each axis-cylinder process divides into an ascending and a descending branch, which run in the dorsal fasciculi. The descending branch runs but a short distance, and ends in the gray matter of the same side of the cord. It gives off a number of collaterals, which also end in the gray matter. The ascending branch may end in the gray matter soon after entering, or it may run in the dorsal fasciculi as far as the medulla, and end in the nuclei of these. In any case it does not cross the middle line. The lower sensory neurone is direct.

The cells about which the axis-cylinder processes and their collaterals of the lower sensory neurone end are of various kinds. They are known as sensory neurones of the second order. In the first place, some of them end about the cell bodies of the lower motor neurones, forming the path for reflexes. They also end about cells whose axis-cylinder processes cross the middle line and run to the opposite side of the brain. In the spinal cord these cells are found in the different parts of the gray matter, and their axis-cylinder processes run in the opposite ventro-lateral ascending tract of Gowers (Fig. 6, 6) and in the ground bundles (fasciculus lateralis proprius and fasciculus ventralis proprius).

In the medulla the nuclei of the dorsal fasciculi (nucleus fasciculi gracilis (Golli) and nucleus fasciculi cuneati (Burdachi)) contain for the most part cells of this character. Their axis-cylinder processes, after crossing, run toward the brain in the medial lemniscus or bundle of the fillet; certain of the longitudinal bundles in the formatio reticularis also represent sensory paths from the spinal cord and medulla toward higher centres. The fibres of the medial lemniscus or fillet do not, however, run directly to the cerebral cortex. They end about cells in the ventro-lateral portion of the optic thalamus, and the tract is continued on by way of another set of neurones, which send processes to end in the cortex of the central convolutions and the parietal lobe. This is the most direct path of sensory conduction,

but by no means the only one. The peripheral sensory neurones may also end about cells in the cord whose axones run but a short distance toward the brain before ending again in the gray matter, and the path, if path it can be called, is made up of a series of these superimposed neurones. The gray matter of the cord itself is also believed to offer paths of sensory conduction. All these paths reach the tegmentum and optic thalamus, and from thence are distributed to the cortex along with the other sensory paths. There may also be paths of sensory conduction through the cerebellum by way of the direct cerebellar tract and Gowers' bundle. From this short summary it is evident that the possible paths of sensory conduction are many, and that our knowledge of them is as yet very indefinite; for this reason disturbances in sensation do not give us as much help in making a local diagnosis as do those of motion. Certain facts are important to keep in mind. The different peripheral nerves contain sensory fibres from definite areas of the skin, and upon this depends the peripheral sensory representation. (See section on Diseases of the Spinal Nerves.)

The sensory areas of the skin are represented in the spinal cord in an entirely different manner from the peripheral representation, just as is the case in regard to motion. The surface of the body has been mapped out into areas which are meant to correspond to the different dorsal roots or spinal segments. In Starr's table the third column indicates his belief. His more recent division of the sensory areas on the limbs is pictured in the American Journal of the Medical Sciences, June, 1895. Figs. 7 and 8 embody the result of Head's work. They are also the areas in which the referred pain and cutaneous tenderness in visceral diseases make their appearance. The cutaneous sensory impressions are in man conducted toward the brain, probably on the opposite side of the cord—that is, the path crosses to the opposite side soon after entering the cord. Muscular sense, on the other hand, is conducted on the same side of the cord in the fasciculi of Goll, to cross above by means of the axones of sensory neurones of the second order in the medulla.

The localization of sensory impressions in the cortex of the brain is not definitely determined, but in a general way it corresponds to the motor representation. Sensation seems, however, to be more widely represented than motion, and to occupy most of the parietal lobe as well as the central convolutions.

The paths for the conduction of the stimuli which underlie the special senses are given in the section upon the cerebral nerves, and it is only necessary here to refer to what is known of the cortical representation of these senses.

Visual impressions are localized in the occipital lobes. The primary visual centre is on the mesial surface in the cuneus, especially about the calcarine fissure, and here are represented the opposite half-visual fields. Some authors believe that there is another higher centre on the outer surface of the occipital lobe, in which the vision of the opposite eye is chiefly represented. However this may be, most authors hold that the angular gyrus of the left hemisphere is a part of the brain in which are stored the memories of the meaning of letters, words, figures, and indeed of all seen

objects. This is designated in the visual speech centre on the diagram (Fig. 3). Flechsig and Monokow do not admit this.

Auditory impressions are localized for the most part in the first temporal convolution and the transverse temporal gyri, and it is in this region in the left hemisphere that the memories of the meanings of heard words and sounds are stored. Musical memories are localized somewhat in front of those for words (Fig. 3). The cortical centres for smell include a part of the base of the frontal lobe, the uncus, and perhaps the gyrus hippocampi. The centres for taste are supposed to be situated near those for smell, but we possess as yet no definite information about them.

Topical Diagnosis.—The successful diagnosis of the position of a lesion in the nervous system depends upon a careful and exhaustive examination into all the symptoms that are present, and then endeavoring with the help of anatomy and physiology to determine the place, a disturbance at which might produce these symptoms.

The abnormalities of motion are usually the most important localizing symptoms, both on account of the ease with which they can be demonstrated, and also because of the comparative accuracy of our knowledge of the motor path.

Lesions in any part of the motor path cause disturbances of motion. If destructive, the function of the part is abolished, and as the result there is *paralysis*. If, on the other hand, the lesion is an irritative one, the structures are thrown into abnormal activity, which produces *abnormal muscular contraction*. The character of the paralysis or of the abnormal muscular contraction varies with lesions of the upper and lower motor segment, the variations depending, first, upon the anatomical position of the two segments; and, secondly, upon the symptoms which are the result of secondary degeneration in each of the segments.

(*a*) **Lesions of the Lower or Spino-muscular Segment.**—*Destructive Lesions.*—It has been stated above that the nutrition of all parts of a neurone depends upon their connection with its healthy cell body; and if the cell body be injured, its processes undergo degeneration, or if a portion of a process be separated from the cell body, that part degenerates along its whole length. This so-called secondary degeneration plays a very important *rôle* in the symptomatology.

In the lower motor segment the degeneration not only affects the axis-cylinder processes which run in the peripheral nerves, but also the muscle fibres in which the axis-cylinder processes end. The degeneration of the nerves and muscles is made evident, first, by the muscles becoming smaller and flabby, and, secondly, by change in their reaction to electrical stimulation. The degenerated nerve gives no response to either the galvanic or the faradic current, and the muscle does not respond to faradic stimulation, but reacts in a characteristic manner to the galvanic current. The contraction, instead of being sharp, quick, lightning-like, as in that of a normal muscle, is slow and lazy, and is often produced by a weaker current, and the anode-closing contraction may be greater than the cathode-closing contraction. This is the reaction of degeneration, but it is not always present in the classical form. The essential feature is the slow, lazy contrac-

tion of the muscle to the galvanic current, and when this is present the muscle is degenerated.

The myotatic irritability, or muscle reflex, and the muscle tonus depend upon the integrity of the reflex arc, of which the lower motor segment is the efferent limb, and in a paralysis due to lesion of this segment the muscle reflexes (tendon reflexes) are abolished and there is a diminished muscular tension.

Lower segment paralyses have for their characteristics degenerative atrophy with the reaction of degeneration in the affected muscles, loss of their reflex excitability, and a diminished muscular tension. These are the general characteristics, but the anatomical relations of this segment also give certain peculiarities in the distribution of the paralyses which help to distinguish them from those which follow lesions of the upper segment, and which also aid in determining the site of the lesion in the lower segment itself. The cell bodies of this segment are distributed in groups, from the level of the peduncles of the brain throughout the whole extent of the spinal cord to its termination opposite the second lumbar vertebra, and their axis-cylinder processes run in the peripheral nerves to every muscle in the body; so that the component parts are more or less widely separated from each other, and a local lesion causes paralysis of only a few muscles or groups of muscles, and not of a whole section of the body, as is the case where lesions affect the upper segment. The muscles which are paralyzed indicate whether the disease is in the peripheral nerves or spinal cord; for, as we have seen above, the muscles are represented differently in the peripheral nerves and in the spinal cord. Sensory symptoms, which may accompany the paralysis, are often of great assistance in making a local diagnosis. Thus, in a paralysis with the characteristics of a lesion of the lower motor segment, if the paralyzed muscles are all supplied by one nerve, and the anæsthetic area of the skin is supplied by that nerve, it is evident that the lesion must be in the nerve itself. On the other hand, if the muscles paralyzed are not supplied by a single nerve, but are represented close together in the spinal cord, and the anæsthetic area corresponds to that section of the cord (see table), it is equally clear that the lesion must be in the cord itself or in its nerve roots.

Irritative Lesions of the Lower Motor Segment.— Lesions of this segment cause comparatively few symptoms of irritation, and our knowledge on the point is neither extensive nor accurate. The fibrillary contractions which are so common in muscles undergoing degeneration are probably due to stimulation of the cell bodies in their slow degeneration, as in progressive muscular atrophy, or to irritation of the axis-cylinder processes in the peripheral nerves, as in neuritis. Lesions which affect the motor roots as they leave the central nervous system may cause spasmodic contractions in the muscles supplied by them. Certain convulsive paroxysms, of which laryngismus stridulus is a type, and to which the spasms of tetany also belong, are believed to be due to abnormal activity in the lower motor centres. These are the "lowest level fits" of Hughlings Jackson. Certain poisons, as strychnia and that of tetanus, act particularly upon these centres.

The principle diseases in which the lower motor segment may be involved are: all diseases involving the peripheral nerves, cerebral and spinal meningitis, injuries, hæmorrhages and tumors of the medulla and cord or their membranes, lesions of the gray matter of the segment, anterior poliomyelitis, progressive muscular atrophy, bulbar paralysis, ophthalmoplegia, syringo-myelia, etc.

(*b*) **Lesions of the Upper Motor Segment.**—*Destructive lesions* cause, as in the lower motor segment, paralysis, and here again the secondary degeneration which follows the lesion gives to the paralysis its distinctive characteristics. In this case the paralysis is accompanied by a spastic condition, shown in an exaggeration of muscle reflex and an increase in the tension of the muscle. It is not accurately known how the degeneration of the pyramidal fibres causes this excess of the muscle reflex. The usual explanation is, that under normal circumstances the upper motor centres are constantly exerting a restraining influence upon the activity of the lower centres, and that when the influence ceases to act, on account of disease of the pyramidal fibres, the latter take on increased activity, which is made manifest by an exaggeration of the muscle reflex.

We have seen that the neurones composing each segment of the motor path are to be considered as nutritional units, and therefore the secondary degeneration in the upper segment stops at the beginning of the lower. For this reason the muscles paralyzed from lesions in the upper segment do not undergo degenerative atrophy, nor do they show any marked change in their electrical reactions.

The separate parts of the upper motor segment lie much more closely together than do those of the lower segment, and therefore a small lesion may cause paralysis in many muscles. This is more particularly true in the internal capsule, where all the axis-cylinder processes of this segment are collected into a compact bundle—the pyramidal tract. A lesion in this region usually causes paralysis of all the muscles on the opposite side of the body—that is, hemiplegia. The pyramidal tract continues in a compact bundle, giving off fibres to the motor nuclei at different levels; a lesion anywhere in its course is followed by paralysis of all the muscles whose nuclei are situated below the lesion. When the disease is above the decussation, the paralysis is on the opposite side of the body; when below, the paralyzed muscles are on the same side as the lesion. Above the internal capsule the path is somewhat more separated, and in the cortex the centres for the movements of the different sections of the body are comparatively far apart, and a sharply localized lesion in this region may cause a more limited paralysis, affecting a limb or a segment of a limb—the cerebral monoplegias; but even here the paralysis is not confined to an individual muscle or group of muscles, as is commonly the case in lower segment paralysis (see Fig. 2 and explanation).

To sum up, the paralyses due to lesions of the upper motor segment are widespread, often hemiplegic; the paralyzed muscles are spastic (the tendon reflexes exaggerated), they do not undergo degenerative atrophy, and they do not present the degenerative reaction to electrical stimulation.

There is an exception to the above statement—that is, in the paralyses

which follow a complete transverse lesion of the spinal cord. Here the limbs are of course completely paralyzed, but instead of being spastic they are flaccid and the deep reflexes are absent. There is, however, no marked atrophy in the muscles, and they react normally to electricity. There is no satisfactory explanation of why the reflexes should be abolished under these conditions.

Irritative Lesions of the Upper Motor Segment.—Our knowledge of such lesions is confined for the most part to those acting on the motor cortex. The abnormal muscular contractions resulting from lesions so situated have as their type the localized convulsive seizures classed under Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex. For instance, such a convulsion, beginning in the muscles of the face, next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—i. e., they destroy the nerve cells of a certain centre, and either in their growth or by their presence throw into abnormal activity those of the surrounding centres.

The upper motor segment is involved in nearly all the diseases of the brain and spinal cord, especially in injuries, tumors, abscesses, and hæmorrhages; transverse lesions of the cord; syringomyelia, progressive muscular atrophy, bulbar paralysis, etc. One lesion often involves both the upper and the lower motor segments, and we have paralysis in the different parts of the body, with the characteristics of each. Such a combination enables us in many cases to make an accurate local diagnosis.

Lesions in the optic path and in the different speech centres also give localizing symptoms, which should be always looked for.

(*c*) **Lesions of the Sensory Path.**—Here again the lesion may be either irritative or destructive. *Irritative lesions* cause abnormal subjective sensory impressions—paræsthesia, formication, a sense of cold or constriction, and pain of every grade of intensity. The character of the sensory symptoms gives very little indication as to the position of the irritating process. Intense pain is, as a rule, a symptom of a lesion in the peripheral sensory neurones, but it may be caused by a disease of the sensory path within the central nervous system.

The exact distribution of symptoms gives us more accurate data, for if they are confined to the distribution of a peripheral nerve or of a spinal segment the indication is plain. If one side of the body is more or less completely affected, we must think of a lesion somewhere within the brain, etc.

Destructive Lesions.—A complete destruction of the sensory paths from any part of the body would of course deprive that part of sensation in all its qualities. This occurs most frequently from injury to the peripheral sensory neurones within the peripheral nerves, and the area of anæsthesia

depends upon the nerve injured. Complete transverse lesion of the cord causes complete anæsthesia below the injury.

Unilateral lesions of the cord, medulla, dorsal part of the pons, tegmentum, thalamus, internal capsule, and cortex cause disturbances of sensation on the opposite side of the body; here again the extent of the defect more than its character helps us to determine the position of the lesion. Hemianæsthesia involving the face as well as the rest of the body can only occur above the place where the sensory paths from the fifth nerve have crossed the middle line on their way to the cortex. This is in the upper part of the pons. From this point to where they leave the internal capsule the sensory paths are in fairly close relation, and are at times involved in a very small lesion. Above the internal capsule the paths diverge quickly, and for this reason only an extensive lesion can involve them all, and in lesions of this part we are more apt to have the sensory disturbances confined to one or the other segments of the body. Unilateral lesions of the pons, medulla, and cord usually cause sensory disturbances on the same side of the body, as well as those on the opposite side. These are due to the involvement of the sensory paths as they enter the central nervous system at or a little below the site of the lesion and before the axones of the sensory neurones of the second order have crossed the middle line. The area of disturbed sensation is limited to the distribution of one or more spinal segments and often indicates accurately the position and extent of the diseased process. As a rule, destructive lesions of the central nervous system do not involve all the paths of sensory conduction, and the loss of sensation is not complete. It is often astonishing how very slight the sensory disturbances are which result from an extensive lesion of the nervous system. Sensation may be diminished in all of its qualities, or, what is more common, certain qualities may be affected while others are normal. These cases of dissociation of sensation, or so-called elective sensory paralysis, have been much studied of late. Thus the sense of pain and temperature may be lost while that of touch remains normal, as is often the case in diseases of the spinal cord, or there may be simply a loss of the muscular sense and of the stereognostic sense (the complex sensory impression which enables one to recognize an object placed in the hand), as occurs frequently from lesions of the cortex. Occasionally pain sensation persists with loss of tactile and thermic sensations. Almost every other combination has been described. It is the distribution more than the character of the sensory defect that is of importance, and often the distribution gives but uncertain indication of the position of the lesion. The combination of the sensory defect with different forms of paralysis gives the most certain diagnostic signs. The student is referred to the sections on the individual parts of the nervous system for a more detailed consideration of the subject.

II. SYSTEM DISEASES.

I. INTRODUCTION.

There are certain diseases of the nervous system which are confined, if not absolutely, still in great part, to definite tracts (combinations of neurones) which subserve like functions. These tracts are called *systems*, and a disease which is confined to one of them is a *system disease*. If more than one system is involved, the process is called a combined system disease. Just what diseases should be classed under these names has given rise to much discussion but to very little agreement. We cannot speak positively; our knowledge is as yet not sufficiently accurate, either in regard to the exact limits of the systems themselves, or to the nature and extent of the morbid process in the several diseases. In the classification which has been adopted in this edition the endeavor has been to make the arrangement as simple as possible, and, while it is based upon what is believed to be the best founded views of the systems and their diseases, there has been no attempt to carry the classification to its logical conclusion, nor have the limits of the theory been always respected.

In general it may be said that the nervous system is composed of two great systems of neurones, the afferent or sensory system and the efferent or motor system, and the connections between them. (See General Introduction.)

Locomotor ataxia is a disease confined to the afferent system, and progressive muscular atrophy is one of the efferent system. Representing typical system diseases as we now understand them, they have been taken as the basis of the classification. Several theories have been advanced to explain why a disease should be limited to a definite system of neurones. One view is based upon the idea that in certain individuals one or the other of these systems has an innate tendency to undergo degeneration; another assumes that neurones with a similar function have a similar chemical construction (which differs from that of neurones with a different function), and this is taken to explain why a poison circulating in the blood should show a selective action for a single functional system of neurones.

In the afferent tract locomotor ataxia stands alone as a system disease. In the efferent tract progressive (central) muscular atrophy is the chief representative, as in it the whole motor path is more or less involved. Theoretically, primary lateral sclerosis is a disease confined to the upper segment of the efferent tract, while anterior poliomyelitis involves the lower segment of the tract.

In connection with progressive (central) muscular atrophy, the other forms of muscular atrophy are considered as a matter of convenience. In other instances, too, diseases are arranged in positions to which they might not be entitled, had a rigid classification of system diseases been maintained.

II. DISEASES OF THE AFFERENT OR SENSORY SYSTEM.

Locomotor Ataxia

(Tabes Dorsalis; Posterior Spinal Sclerosis).

Definition.—An affection characterized clinically by incoördination, sensory and trophic disturbances, and involvement of the special senses, particularly the eyes. Anatomically there are found degeneration of the posterior roots and of the dorsal columns of the cord; sometimes the spinal ganglia and peripheral nerves are affected. Foci of degeneration in the basal ganglia and degenerative changes in the cortex cerebri have been described.

Etiology.—It is a widespread disease, more frequent in cities than in the country. The relative proportion may be judged from the fact that of 8,642 cases in the neurological dispensary of the Johns Hopkins Hospital there were 89 cases of locomotor ataxia (H. M. Thomas). Males are attacked more frequently than females, the proportion being at least 10 to 1. Mitchell has called attention to the fact that it is a rare disease in the negro. It is a disease of adult life, a majority of the cases occurring between the thirtieth and fortieth years. Occasionally cases are seen in young men. The form of ataxia which occurs in children is a different disease. Of special causes syphilis is the most important. According to the figures of Erb, Fournier, and Gowers, in from 50 to 75 per cent of all cases there is a history of this disease. Erb's recent figures are most striking; of 300 cases of tabes in private practice 89 per cent had had syphilis. Moebius goes so far as to say, "The longer I reflect upon it, the more firmly I believe that tabes never originates without syphilis."

Excessive fatigue, overexertion, injury, exposure to cold and wet, and sexual excesses are all assigned as causes. There are instances in which the disease has closely followed severe exposure. James Stewart has noted that the Ottawa lumbermen, who live a very hard life in the camps during the winter months, are frequently the subjects of locomotor ataxia. Trauma has been noted in a few cases. Alcoholic excess does not seem to predispose to the disease. Among patients in the better classes of life I do not remember one in which there had been a previous history of prolonged drunkenness. There are now a good many cases on record of the existence of the disease in both husband and wife.

Morbid Anatomy and Pathology.—Our conception of tabes dorsalis has undergone radical alteration, and the studies of Leyden, Redlich, Marie, and others have shown that it can no longer be regarded as a primary sclerosis of the dorsal columns. These, it will be remembered, are made up, in great part, of the axis-cylinder processes of the spinal ganglia, and they, with their branches, represent in the cord the paths of sensory conduction. The peripheral sensory nerves represent the protoplasmic processes of the spinal ganglia, which important structures are the trophic centres both for the sensory nerves as well as for the axis-cylinder processes which make up the dorsal columns of the cord. Marie calls attention also

to the possibility of the existence of peripheral or terminal ganglion cells which are found in different organs—cells from which certain of the sensory fibres are derived which go to form the dorsal nerve-roots. According to the general laws of nerve physiology, already mentioned, lesions of the nerve ganglia would be followed by degeneration of the dorsal root-fibres and of their continuation in the cord, and this is practically what the recent theory of tabes involves. The changes in the dorsal columns are merely a sequence, and not the primary disease. The fibres of the dorsal root are divided into three sets:

(1) The short fibres, which pass almost directly into the dorsal cornu after entering the cord.

(2) Fibres of moderate length, which run upward in the cord; some of them enter the dorsal horn at its middle part, while others pass into Clarke's column. The fibres of this group run in the fasciculus cuneatus of Burdach.

(3) A group of long fibres, which are derived chiefly from the roots of the cauda equina, and which pass the whole length of the cord to enter certain nuclei in the medulla. They form the fasciculus gracilis of Goll.

The initial cord lesion in tabes is found in the dorsal root-zone and in the zone or tract of Lissauer, a narrow portion situated between the margin of the cord and the apex of the posterior horn. In the fasciculus of Burdach the sclerosis is in almost direct proportion to the duration of the disease, slight at first and centrally placed, and becoming widespread as the disease advances. The fasciculus of Goll is affected slightly in the early stages, but in the advanced stage there is extensive sclerosis. Marie correlates the sclerosis of these different parts with the different groups of nerve-fibres of the dorsal root, the dorsal root-zone and the zone of Lissauer degenerating from the involvement of the short fibres; the sclerosis of the fasciculi of Burdach and the disappearance of the network of the nerve-fibres in the column of Clarke being due to the degeneration of the second group, the fibres of moderate length; while the sclerosis of the fasciculi of Goll is caused by the degeneration of the third group, namely, the long fibres. He suggests also that groups of fibres in the different dorsal roots are not simultaneously affected, and the lesions may be in an advanced stage in one region and but slight in the other. "*The lesions of the spinal cord in tabes occur by segments*, each dorsal root bringing into the dorsal column a fresh contingent of degenerated fibres."

According to this interesting hypothesis the lesions of the ganglia of the dorsal roots are responsible, in part at least, for the peripheral neuritis, since in degeneration of the spinal ganglia and consequent loss of trophic influence there would necessarily be degeneration in the peripheral nerve-trunks. Possibly, too, Marie suggests, the degeneration of the *peripheral* ganglion cells may have a good deal to do with the neuritis of tabes.

Obersteiner and Redlich, while agreeing that the degeneration of the dorsal columns of the cord is dependent upon a disease in the dorsal roots, believe, at least for most cases, that the change in the latter is secondary to a chronic inflammation of the pia mater, which, by making pressure on the

dorsal root-fibres just where they are poor in myeline, causes them to degenerate.

The spinal ganglia have been found diseased in certain cases, but in other cases no change whatever could be detected, even by the aid of the most delicate technique, and Marie acknowledges that there is very little anatomical proof for his theory that it is these structures that are primarily affected in tabes.

Trepinski has divided the dorsal fasciculi into different systems according to the time of the development of their myeline, and has endeavored to show that the sclerosis in tabes follows these systems.

Symptoms.—These are best considered under three stages—the incipient stage, the ataxic stage, and the paralytic stage.

The Incipient Stage.—This is sometimes called the preataxic stage. The manner in which tabes makes its onset differs very widely in the different cases, and mistakes in diagnosis are often made early in the disease. The following are the most characteristic initial symptoms:

Pains, usually of a sharp stabbing character; hence the term lightning pains. They last for only a second or two and are most common in the legs. They may be associated with a hot burning feeling. Occasionally herpes may develop at the site of the pain. They may occur at irregular intervals, and are more prone to follow excesses or to come on when health is impaired. The gastric crises and other crises may occur in the disease. Paræsthesia may also be among the first symptoms. Numbness of the feet, tingling, etc., and at times a sense of constriction about the body.

Ocular Symptoms.—(*a*) Optic atrophy. This occurs in about 10 per cent of the cases, and is often an early and even the first symptom. There is a gradual loss of vision, which in a large majority of cases leads to total blindness. (*b*) Ptosis, which may be double or single. (*c*) Paralysis of the external muscles of the eye. This may be of a single muscle or occasionally of all of the muscles of the eye. The paralysis is often transient, the patient merely complaining that he saw double for a certain period. (*d*) Argyll Robertson pupil, in which there is loss of the iris reflex to light but contraction during accommodation. The pupils are very small—spinal myosis.

Bladder Symptoms.—The first warning of the disease which the patient has may be a certain difficulty in emptying the bladder. Incontinence of urine occurs only at a later stage of the disease. Decrease in sexual desire and power may also be an early symptom.

Trophic Disturbances.—These usually occur later in the disease, but at times they are very early symptoms and it is not very infrequent to have one's attention called to the trouble by the presence of a perforating ulcer or of a characteristic Charcot's joint.

Loss of the Knee-jerk.—This early and most important symptom may occur years before the development of ataxia. Even alone it is of great moment, since it is very rare to meet with individuals in whom the knee-jerk is normally absent. The combination of loss of the knee-kick with one or more of the symptoms mentioned above, especially with the lightning pains and ptosis or Argyll Robertson pupil, is practically diagnostic. The

knee-jerk is not lost suddenly, but gradually decreases, often disappearing in one leg before the other.

These are the most common symptoms of the initial stage of tabes and may persist for years without the development of incoördination. The patient may look well and feel well, and be troubled only by occasional attacks of lightning pains or of one of the other subjective symptoms. Moebius goes so far as to state that the typical Argyll Robertson pupil means either tabes or general paralysis, and that paralysis of the external muscles of the eye developing in adults are of almost equal importance, especially if they develop painlessly.

The time between the syphilitic infection and the occurrence of the first symptoms of locomotor ataxia varies within wide limits. About one half the cases occur between the sixth and fifteenth year, but many begin even later than this.

The disease may never progress beyond this stage, and when optic atrophy develops early and leads to blindness, ataxia rarely, if ever, supervenes. There is a sort of antagonism between the ocular symptoms and the progress of the ataxia. Charcot laid considerable stress upon this, and both Dejerine and Spiller have since emphasized the point.

Ataxic Stage.—*Motor Symptoms.*—The ataxia is believed to be due to a disturbance or loss of the afferent impulses from the muscles, and a disturbance of the muscle sense itself can usually be demonstrated. It develops gradually. One of the first indications to the patient is inability to get about readily in the dark or to maintain his equilibrium when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position, and he may be quite unable to stand on one leg. This is known as Romberg's symptom. He does not start off promptly at the word of command. On turning quickly he is apt to fall. He descends stairs with more difficulty than he ascends them. Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick, the eyes are directed to the ground, the body is thrown forward, and the legs are wide apart. In walking, the leg is thrown out violently, the foot is raised too high and is brought down in a stamping manner with the heel first, or the whole sole comes in contact with the ground. Ultimately the patient may be unable to walk without the assistance of two canes. This gait is very characteristic, and unlike that seen in any other disease. The incoördination is not only in walking, but in the performance of other movements. If the patient is asked, when in the recumbent posture, to touch the knee with one foot, the irregularity in the movement is very evident. Incoördination of the arms is less common, but usually develops in some grade. It may in rare instances exist before the incoördination of the legs. It may be tested by asking the patient to close his eyes and to touch the tip of the nose or the tip of the ear with the finger, or with the arms thrust out to bring the tips of the fingers together. The incoördination may early be noticed by a difficulty which the patient experiences in buttoning his collar or in performing one of the ordinary routine acts of dressing.

One of the most striking features of the disease is that with marked

incoördination there is no loss of muscular power. The grip of the hands may be strong and firm, the power of the legs, tested by trying to flex them, may be unimpaired, and their nutrition, except toward the close, may be unaffected.

There is a remarkable muscular relaxation which enables the joints to be placed in positions of hyperextension and hyperflexion. It gives sometimes a marked backward curve to the legs. Fränkel, who calls the condition hypotonia, says it may be an early symptom.

Sensory Symptoms.—The lightning pains may persist. They vary greatly in different cases. Some patients are rendered miserable by the frequent occurrence of the attacks; others escape altogther. In addition, common symptoms are tingling, pins and needles, particularly in the feet, and areas of hyperæsthesia or of anæsthesia. The patient may complain of a change in the sensation in the soles of the feet, as if cotton was interposed between the floor and the skin. Sensory disturbances occur less frequently in the hands. Objective sensory disturbances can usually be demonstrated, and indeed almost every variety of sensory disturbance has been described. They have been carefully studied in this country by Knapp and by Patrick, and in Europe by many observers. Bands about the chest of a moderate grade of anæsthesia are not uncommon; they are apt to follow the distribution of spinal segments. The most marked disturbances are usually found on the legs. Retardation of the sense of pain is common, and a pin-prick on the foot is first felt as a simple tactile impression, and the sense of pain is not perceived for a second or two or may be delayed for as much as ten seconds. The pain felt may persist. A curious phenomenon is the loss of the power of localizing the pain. For instance, if the patient is pricked on one limb he may say that he feels it on the other (allocheiria), or a pin-prick on the foot may be felt on both feet. The muscular sense which is usually affected early, becomes much impaired and the patient no longer recognizes the position in which his limbs are placed. This may be present in the pre-ataxic stage.

Reflexes.—As mentioned, the loss of the knee-jerk is one of the earliest symptoms of the disease. Occasionally a case is found in which it is retained. The skin reflexes may at first be increased, but later are usually involved with the deep reflexes.

Special Senses.—The eye symptoms noted above may be present, but, as mentioned, ataxia is rare with atrophy of the optic nerve.

Deafness may develop, due to lesion of the auditory nerve. There may also be attacks of vertigo. Olfactory symptoms are rare.

Visceral Symptoms.—Among the most remarkable sensory disturbances are the tabetic crises, severe paroxysms of pain referred to various viscera; thus laryngeal, gastric, nephric, rectal, urethral, and clitoral crises have been described. The most common are the gastric and laryngeal. In the former there are intense pains in the stomach, vomiting, and a secretion of hyperacid gastric juice. The attack may last for several days or even longer. There may be severe pain without any vomiting. The attacks are of variable intensity and usually require morphia. Paroxysms of rectal pain and tenesmus are described. They have not been common in my

experience. Laryngeal crises also are rare. There may be true spasm with dyspnœa and noisy inspiration. In one instance at least the patient has died in the attack.

The sphincters are frequently involved. Early in the disease there may be a retardation or hesitancy in making water. Later there is retention, and cystitis may occur. Unless great care is taken the inflammation may extend to the kidneys. Constipation is extremely common. Late in the disease the sphincter ani is weakened. The sexual power is usually lost in the ataxic stage.

Trophic Changes.—Skin rashes may develop in the course of the lightning pains, such as herpes, œdema, or local sweating. Alteration in the nails may occur. A perforating ulcer may develop on the foot, usually beneath the great toe. A perforating buccal ulcer has also been described. Onychia may prove very troublesome.

The arthropathies or joint lesions affect chiefly the knees. They are unquestionably associated with the disease itself, and are not necessarily a result of trauma. The condition, known as Charcot's joint, is anatomically similar to that of chronic arthritis deformans. The effusion may be rapid and there may be great disintegration and destruction of the cartilages and bones, leading to dislocation and deformity. Suppuration may occur. Spontaneous fractures may occur. Among other trophic disturbances may be mentioned atrophy of the muscles, which is usually a late manifestation, but may be localized and associated with neuritis. In any very large collection of cases many instances of atrophy are found, due either to involvement of the ventral horns or to peripheral neuritis.

Cerebral Symptoms.—Hemiplegia may develop at any stage of the disease, more commonly when it is well advanced. It may be due to hæmorrhagic softening in consequence of disease of the vessels or to progressive cortical changes. Hemianæsthesia is sometimes present. Very rarely the hemiplegia is due to coarse syphilitic disease.

Dementia paralytica frequently exists with tabes, and it may be extremely difficult to determine which has been the primary affection; indeed, some authors believe that these two diseases are simply different localizations of the same morbid process. In a majority of the cases the symptoms of locomotor ataxia have preceded those of general paresis. In other instances melancholia, dementia, or paranoia develop.

(*c*) **Paralytic Stage.**—After persisting for an indefinite number of years the patient gradually loses the power of walking and becomes bedridden or paralyzed. In this condition he is very likely to be carried off by some intercurrent affection, such as pyelo-nephritis, pneumonia, or tuberculosis.

The Course of the Disease.—A patient may remain in the pre-ataxic stage for an indefinite period; and the loss of knee-jerk and the gray atrophy of the optic nerves may be the sole indication of the true nature of the disease. In such cases incoördination rarely develops. In a majority of cases the progress is slow, and after six or eight years, sometimes less, the ataxia is well developed. The symptoms may vary a good deal; thus the pains, which may have been excessive at first, often lessen. The disease may remain stationary for years; then exacerbations occur and it

makes rapid progress. Occasionally the process seems to be arrested. There are instances of what may be called acute ataxia, in which, within a year or even less, the incoördination is marked, and the paralytic stage may develop within a few months. The disease itself rarely causes death, and after becoming bedridden the patient may live for fifteen or twenty years.

Diagnosis.—In the initial stage the combination of lightning pains and the absence of knee-jerk is distinctive. The association of progressive atrophy of the optic nerves with loss of knee-jerk is also characteristic. The early ocular palsies are of the greatest importance. A squint, ptosis, or the Argyll Robertson pupil may be the first symptom, and may exist with the loss only of the knee-jerk. Loss of the knee-jerk alone, however, does occasionally occur in healthy individuals. A history of preceding syphilis lends added weight to the symptoms, and its presence or absence may be of the utmost importance in determining the diagnosis. If the possibility of syphilitic infection can be excluded, a circumstance but too rarely met with, only the most unequivocal combination of symptoms can justify the diagnosis of locomotor ataxia.

The diseases most likely to be confounded with locomotor ataxia are: (1) *Peripheral Neuritis.*—The steppage gait of arsenical, alcoholic, or diabetic paralysis is quite unlike that of locomotor ataxia. In these forms there is a paralysis of the feet and the leg is lifted high in order that the toes may clear the floor. The use of the word tabes in this connection should no longer be continued. In the rare cases in which the muscle sense nerves are particularly affected and in which there is true ataxia, the absence of the lightning pains and eye symptoms and the history will suffice in the majority of cases to make the diagnosis clear. In diphtheritic paralysis the early loss of the knee-jerk and the associated eye symptoms may suggest tabes, but the history, the existence of paralysis of the throat, and the absence of pains render a diagnosis easy.

(2) *Ataxic Paraplegia.*—Marked incoördination with spastic paralysis is characteristic of the condition which Gowers has termed ataxic paraplegia. In a majority of the cases this affection is distinguished also by the absence of pains and of eye symptoms.

(3) *Cerebral Disease.*—In diseases of the brain involving the afferent tracts ataxia is at times a prominent symptom. It is usually unilateral or limited to one limb; this, with the history and the associated symptoms, excludes tabes.

(4) *Cerebellar Disease.*—The cerebellar incoördination has only a superficial resemblance to that of locomotor ataxia, and is more a disturbance of equilibrium than a true ataxia; the knee-jerk is usually present, there are no lightning pains, no sensory disturbances; while, on the other hand, there are headache, optic neuritis, and vomiting.

(5) Some *acute affections* involving the dorsal columns of the cord may be followed by incoördination and resemble tabes very closely. In a case under my care, the gait was characteristic and Romberg's symptom was present. The knee-jerk, however, was retained and there were no ocular symptoms. The condition had developed within three or four months, and

there was a well-marked history of syphilis. Under large doses of iodide of potassium the ataxia and other symptoms completely disappeared.

(6) *General Paresis.*—In some cases this offers a serious difficulty. In the first place, in general paresis, tabetic symptoms often develop; on the other hand, there are cases of locomotor ataxia in which, toward the end, there are symptoms of general paresis. Cases of unusually acute ataxia with mental symptoms belong, as a rule, to the former disease. The question will be considered under general paresis.

(7) Visceral crises and neuralgic symptoms may lead to error, and in middle-aged men with severe, recurring attacks of gastralgia it is always well to bear in mind the possibility of tabes, and to make a careful examination of the eyes and of the knee-jerk.

Prognosis.—Complete recovery cannot be expected, but arrest of the process is not uncommon and a marked amelioration of the symptoms is frequent. Optic-nerve atrophy, one of the most serious events in the disease, has this hopeful aspect—that incoördination rarely follows and the progress may be arrested. The optic atrophy itself is occasionally checked. On the whole, the prognosis in tabes is bad. The experience of such men as Weir Mitchell, Charcot, and Gowers is distinctly opposed to the belief that locomotor ataxia is ever completely cured.* No such instance has come under my personal observation.

Treatment.—To arrest the progress and to relieve, if possible, the symptoms are the objects which the practitioner should have in view. A quiet, well-regulated method of life is essential. It is not well, as a rule, for a patient to give up his occupation so long as he is able to keep about and perform ordinary work. I know tabetics who have for years conducted large businesses, and there have been several notable instances in our profession of men who have risen to distinction in spite of the existence of this disease. Excesses of all sorts, more particularly *in baccho et venere*, should be carefully avoided. A man in the pre-ataxic stage should not marry.

Care should be taken in the diet, particularly if gastric crises have occurred. To secure arrest of the disease many remedies have been employed. Although syphilis plays such an important *rôle* in the etiology, it is universally acknowledged that neither mercury nor the iodide of potassium have as a rule the slightest influence over the tabetic lesions. To this there is but one exception—when the syphilis is comparatively recent; when the symptoms develop within two years of the primary infection, there is then a possibility of arrest by mercury and iodide of potassium. However, they do not always relieve. In two cases of very rapidly progressing tabes following syphilis this medication was of no avail. Of remedies which may be tried and are believed by some writers to retard the progress, the following are recommended: Arsenic in full doses, nitrate of silver in quarter-grain doses, Calabar bean, ergot, and the preparations of gold.

The treatment by suspension introduced a few years ago has already been practically abandoned. Good effects certainly have followed in a few

* For a study of reputed cures, see L. C. Gray, N. Y. Medical Journal, November, 1889.

cases, but it was unreasonable from the outset, either on therapeutic or scientific grounds, to hope that by such a measure permanent changes could be induced in the pathological condition. The benefits were due in great part to suggestion and to psychical effects. In any case it must be used with caution.

For the pains, complete rest in bed, as advised by Weir Mitchell, and counter-irritation to the spine (either blisters or the thermo-cautery) may be employed. The severe spells which come on particularly after excesses of any kind are often promptly relieved by a hot bath or by a Turkish bath. A prolonged course of nitrate of silver seems in some cases to allay the pains and lessen the liability to the attacks. I have never seen ill effects from its use in spinal sclerosis. Antipyrin and antifebrin may be employed, and occasionally do good, but their analgesic powers in this disease have been greatly overrated. Cannabis indica is sometimes useful. In the severe paroxysms of pain hypodermics of morphia or of cocaine must be used. The use of morphia should be postponed as long as possible. Electricity is of very little benefit. For the severe attacks of gastralgia, morphia is also required. The laryngeal crises are rarely dangerous. An application of cocaine may be made during the spasm, or a few whiffs of chloroform may be given, or nitrite of amyl. In all cases of tabes with increased arterial tension the prolonged use of nitroglycerin, given in increasing doses until the physiological effect is produced, is of great service in allaying the neuralgic pains and diminishing the frequency of the crises. Its use must be guarded when there is aortic insufficiency. The special indication is increased tension. The bladder symptoms demand constant care. When the organ cannot be perfectly emptied the catheter should be used, and the patient may be taught its use and how to keep it thoroughly sterilized.

Fränkel's method of re-education often helps the patient to regain to a considerable extent the control of the voluntary movements which he has lost. By this method the patient is first taught, by repeated systematic efforts, to perform simple movements; from this he goes to more and more complex movements. The treatment should be directed and supervised by a trained teacher, as the result depends upon the skill of the teacher quite as much as upon the perseverance of the patient.

III. DISEASES OF THE EFFERENT OR MOTOR TRACT.

A. OF THE WHOLE TRACT.

1. Progressive (Central) Muscular Atrophy

(*Poliomyelitis Anterior Chronica; Amyotrophic Lateral Sclerosis; Progressive Bulbar Paralysis*).

Definition.—A disease characterized by a chronic degeneration of the motor tract. The whole tract is usually involved, but at times the degeneration is limited to the lower segments. Associated with it is a progressive atrophy of the muscles, combined with more or less spastic rigidity.

Three affections, as a rule described apart, belong together in this category: (*a*) Progressive muscular atrophy of spinal origin; (*b*) amyotrophic lateral sclerosis; and (*c*) progressive bulbar paralysis. A slow atrophic change in the motor neurones is the anatomical basis, and the disease is one of the whole motor path, involving, in many cases, the cortical, bulbar, and spinal centres. There may be simple muscular atrophy with little or no spasm, or progressive wasting with marked spasm and great increase in the reflexes. In others, there are added symptoms of involvement of the motor nuclei in the medulla—a glosso-labio-laryngeal paralysis; while in others, again, with atrophy (especially of the arms), a spastic condition of the legs and bulbar phenomena, tremors develop and signs of cortical lesion. These various stages may be traced in the same case.

For convenience, bulbar paralysis will be considered separately, and I shall here take up together *progressive muscular atrophy* and *amyotrophic lateral sclerosis.*

The disease is known as the Aran-Duchenne type of progressive muscular atrophy and as Cruveilhier's palsy, after the French physicians who early described it. Lewy and Lockhart Clarke first demonstrated that the cells of the ventral horns of the spinal cord were diseased. Charcot separated two types —one with simple wasting of the muscles, due, he believed, to degeneration confined to the ventral horns (and to this he restricted the name progressive muscular atrophy—type, Aran-Duchenne); the other, in which there was spastic paralysis of the muscles followed by atrophy. As the anatomical basis for this he assumed a primary degeneration of the pyramidal tracts and a secondary atrophy of the ventral horns. To this he gave the name of amyotrophic lateral sclerosis. There is but little evidence, however, to show that any such sharp distinction can be made between these two diseases, and Leyden and Gowers regard them as identical.

Etiology.—The cause of the disease is unknown. It is more frequent in males than in females. It affects adults, developing after the thirtieth year, though occasionally younger persons are attacked. A large majority of all cases of progressive muscular atrophy under twenty-five years of age belong to the dystrophies. Cold, wet, exposure, fright, and mental worries are mentioned as possible causes. Erb has lately called attention to certain cases following injury. Hereditary influences are present in certain cases. The rare form which occurs in infancy usually affects several members of the same family. Hereditary and family influences, however, play but a small part in the etiology of this disease, and in this it is in contrast to progressive neural muscular atrophy and the dystrophies. Yet, in the Farr family, which I recorded some years ago, in which thirteen members were affected in two generations, with the exception of two, the cases occurred or proved fatal above the age of forty, and the late onset speaks rather for a central affection. The spastic form may develop late in life—after seventy—as a senile change.

Morbid Anatomy.—The essential anatomical change is a slow degeneration of the motor path, involving particularly the lower motor neurones. The upper neurones are also involved, either first, simultaneously, or at a later period. Associated with the degeneration in the cells of the

ventral horns there is a degenerative atrophy of the muscles. The following are the important anatomical changes: (*a*) The gray matter of the cord shows the most marked alteration. The large ganglion cells of the ventral horns are atrophied, or, in places, have entirely disappeared, the neuroglia is increased, and the medullated fibres are much decreased. The fibres of the ventral nerve-roots passing through the white matter are wasted. (*b*) The ventral roots outside of the cord are also atrophied. (*c*) The muscles which are affected show degenerative atrophy, and the inter-muscular branches of the motor nerve are degenerated. (*d*) The degeneration of the gray matter is rarely confined to the cord, but extends to the medulla, where the nuclei of the motor cerebral nerves are found extensively wasted. (*e*) In a majority of all the cases there is sclerosis in the ventro-lateral white tracts, the lateral pyramidal tracts particularly are diseased, but the degeneration is not confined to these tracts, and extends into the ventro-lateral ground bundles. The direct cerebellar and the ventro-lateral ascending tracts are spared. The degeneration in the pyramidal tracts extends toward the brain to different levels, and in several cases has been traced to the motor cortex, the cells of which have been found degenerated. In the medulla the medial longitudinal fasciculus has been found diseased. (*f*) In those cases in which no sclerosis has been found in the pyramidal tracts there has been a sclerosis of the ventro-lateral ground bundle (short tracts).

Symptoms.—Irregular pains may precede the onset of the wasting, and cases may be treated for chronic rheumatism. The hands are usually first affected, and there is difficulty in performing delicate manipulations. The muscles of the ball of the thumb waste early, then the interossei and lumbricales, leaving marked depressions between the metacarpal bones. Ultimately the contraction of the flexor and extensor muscles and the extreme atrophy of the thumb muscles, the interossei, and lumbricales produces the claw-hand—*main en griffe* of Duchenne. The flexors or the forearm are usually involved before the extensors. In the shoulder-girdle the deltoid is first affected; it may waste even before the other muscles of the upper extremity. The trunk muscles are gradually attacked; the upper part of the trapezius long remains unaffected. Owing to the feebleness of the muscles which support it, the head tends to fall forward. The platysma myoides is unaffected and often hypertrophies. The arms and the trunk muscles may be much atrophied before the legs are attacked. The face muscles are attacked late. Ultimately the intercostal and abdominal muscles may be involved, the wasting proceeds to an extreme grade, and the patient may be actually "skin and bone," and, as "living skeletons," the cases are not uncommon in "museums" and "side-shows." Deformities and contractures result, and lordosis is almost always present. A curious twitching of the muscles (fibrillation) is a common symptom, and may occur in muscles which are not yet attacked. It is a most important symptom, but is not, as was formerly supposed, a characteristic feature of the disease. The irritability of the muscles is increased. Sensation is unimpaired, but the patient may complain of numbness and coldness of the affected limbs. The galvanic and faradic irritability of the muscles progressively dimin-

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In some acute cases there is a good deal of
muscular pain.

ishes and may become extinct, the galvanic persisting for the longer time. In cases of rapid wasting and paralysis there may be the reaction of degeneration. The excitability of the nerve-trunks may persist after the muscles have ceased to respond. The loss of power is usually proportionate to the wasting.

The foregoing description applies to the group of cases in which the atrophy and paralysis are flaccid—*atonic*, as Gowers calls it. In other cases, those which Charcot describes as amyotrophic lateral sclerosis, spastic paralysis precedes the wasting. This *tonic* atrophy first involves the arms and then the legs. The reflexes are greatly increased. It is one of the rare conditions in which a jaw clonus may be obtained. The most typical condition of spastic paraplegia may be produced. On starting to walk, the patient seems glued to the ground and makes ineffectual attempts to lift the toes; then four or five short, quick steps are taken on the toes with the body thrown forward; and finally he starts off, sometimes with great rapidity. Some of the patients can walk up and down stairs better than on the level. The wasting is never so extreme as in the atonic form, and the loss of power may be out of proportion to it. The sphincters are unaffected. Sexual power may be lost early. Cases are met with which correspond accurately to the clinical picture given by Charcot of amyotrophic lateral sclerosis. These are not very common, and it is much more usual to have a combination of the two types. A flaccid atrophic paralysis with increased reflexes is often met with. These differences depend upon the relative extent of the involvement of the upper and lower motor segments and the time of the involvement of each.

As the degeneration extends upward an important change takes place from the development of bulbar symptoms, which may, however, precede the spinal manifestations. The lips, tongue, face, pharynx, and larynx may be involved. The lips may be affected and articulation impaired for years before serious symptoms occur. In the final stage there may be tremor, the memory fails, and a condition of dementia may develop.

Gowers gives the following useful classification of the varieties of this affection: (1) Atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting or with only slight wasting; and (3) atonic atrophy, rarely extreme in degree, with exaggeration of the reflexes. These conditions may "coexist in every degree and combination—between universal atonic atrophy on the one hand and universal spastic paralysis without wasting on the other."

Diagnosis.—Progressive (central) muscular atrophy begins, as a rule, in adult life, without hereditary or family influences (the early infantile form being an exception), and usually affects first the muscles of the thumb, and gradually involves the interossei and lumbricales. Fibrillary contractions are common, electrical changes occur, and the deep reflexes are usually increased. These characteristics are usually sufficient to distinguish it from the other forms of muscular wasting.

In syringo-myelia the symptoms may be very similar to those in the spastic form of muscular atrophy. The sensory disturbances in the former disease make, as a rule, the diagnosis clear, but when these are absent or

but little developed it may be very difficult or even impossible to distinguish the diseases.

Treatment.—The disease is incurable. I have never seen the slightest benefit from drugs or electricity. The downward progress is slow but certain, though in a few cases a temporary arrest may take place. With a history of syphilis, mercury and iodide of potassium may be tried, and Gowers recommends courses of arsenic and the hypodermic injection of strychnine. Probably the most useful means is systematic massage, particularly in the spastic cases.

Bulbar Paralysis (Glosso-labio-laryngeal Paralysis).

When the disease affects the motor nuclei of the medulla first or early, it is called bulbar paralysis, but it has practically no independent existence, as the spinal cord is sooner or later involved.

Symptoms.—The disease usually begins with slight defect in the speech, and the patient has difficulty in pronouncing the dentals and linguals. The paralysis starts in the tongue, and the superior lingual muscle gradually becomes atrophied, and finally the mucous membrane is thrown into transverse folds. In the process of wasting the fibrillary tremors are seen. Owing to the loss of power in the tongue, the food is with difficulty pushed back into the pharynx. The saliva also may be increased, and is apt to accumulate in the mouth. When the lips become involved the patient can neither whistle nor pronounce the labial consonants. The mouth looks large, the lips are prominent, and there is constant drooling. The food is masticated with difficulty. Swallowing becomes difficult, owing partly to the regurgitation into the nostrils, partly to the involvement of the pharyngeal muscles. The muscles of the vocal cords waste and the voice becomes feeble, but the laryngeal paralysis is rarely so extreme as that of the lips and tongue.

The course of the disease is slow but progressive. Death often results from an aspiration pneumonia, sometimes from choking, more rarely from involvement of the respiratory centres. The mind usually remains clear. The patient may become emotional. In a majority of the cases the disease is only part of a progressive atrophy, either simple or associated with a spastic condition. In the latter stage of amyotrophic lateral sclerosis the bulbar lesions may paralyze the lips long before the pharynx or larynx becomes affected.

The *diagnosis* of the disease is readily made, either in the acute or chronic form. The involvement of the lips and tongue is usually well marked, while that of the palate may be long deferred. A condition has been described, however, which may closely simulate bulbar paralysis. This is the so-called *pseudo-bulbar* form or bulbar palsy of cerebral origin. Bilateral disease of the motor cortex in the lower part of the ascending frontal convolution, or about the knee of the internal capsule, may cause paralysis of the lips and tongue and pharynx, which closely simulates a lesion of the medulla. Sometimes the symptoms appear on one side, but in many instances they develop suddenly on both sides. A bilateral le-

sion has usually been found, but in several instances the disease was unilateral.

The so-called *acute bulbar paralysis* may be due to (*a*) hæmorrhagic or embolic softening in the pons and medulla; (*b*) acute inflammatory softening, analogous to polio-myelitis, occurring occasionally as a post-febrile affection. It usually comes on very suddenly, hence the term apoplectiform. The symptoms in this form may correspond closely to those of an advanced case of chronic bulbar paralysis. The sudden onset and the associated symptoms make the diagnosis easy. In these acute cases there may be loss of power in one arm, or hemiplegia, sometimes alternate hemiplegia, with paralysis on one side of the face and loss of power on the other side of the body.

2. Progressive Neural Muscular Atrophy.

This form, known also as the peroneal type, or by the names of the men who have described it most accurately of late—namely, Charcot, Marie, and Tooth—occurs either as a hereditary or as a family affection. It usually begins in early childhood, affecting first the muscles of the feet and the peroneal group; as a result of the weakening of these muscles, club-foot, either pes equinus or pes equino-varus occurs. In rare instances the disease may begin in the hands, but the upper limbs, as a rule, are not affected for some years after the legs are attacked, and the trouble then begins in the small muscles of the hands. Sensory disturbances are frequently present and form important diagnostic features. Fibrillary contractions and twitchings also occur. The electrical reactions are altered; there is either a loss or a very great decrease of the excitability, which can be demonstrated not only in the atrophic muscles, but also in muscles and nerves which are apparently normal.

This form of muscular atrophy seems to stand between the central form and the muscular dystrophies. Occurring in families and beginning in early life, it resembles the latter, but it is more like the former in that fibrillary contractions and muscular twitchings are common, that the small muscles of the hand are apt to be involved, and that electrical changes are present. In the prominence of sensory symptoms it differs from both. In cases of acquired double club-foot this disease should be suspected.

3. The Muscular Dystrophies

(*Dystrophia muscularis progressiva*, Erb).

Definition.—Muscular wasting, with or without an initial hypertrophy, beginning in various groups of muscles, usually progressive in character, and dependent on primary changes in the muscles themselves. A marked hereditary disposition is met with in the disease.

Etiology.—No etiological factors of any moment are known other than heredity. The influence may show itself by true heredity—the disease occurring in two or more generations—or several members of the same generation may be affected, showing a family tendency. Many members of the same family may be attacked through several generations. Males,

as a rule, are more frequently affected than females. The diseas is usually transmitted through the mother, though she may not herself be affected. As many as 20 or 30 cases have been described in five generations. In Erb's cases 44 per cent showed no heredity. The disease usually sets in before puberty, but may be as late as the twentieth or twenty-fifth year, or in some instances even later.

Symptoms.—The first symptom noticed is, as a rule, clumsiness in the movements of the child, and on examination certain muscles or groups of muscles seem to be enlarged, particularly those of the calves. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps and infraspinatus, are the next most frequently involved, and may stand out with great prominence. The muscles of the neck, face, and forearm rarely suffer. Sometimes only a portion of a muscle is involved. With this hypertrophy of some muscles there is wasting of others, particularly the lower portion of the pectorals and the latissimus dorsi. The attitude when standing is very characteristic. The legs are far apart, the shoulders thrown back, the spine is greatly curved, and the abdomen protrudes. The gait is waddling and awkward. In getting up from the floor the position assumed, so well known now through Gowers' figures, is pathognomonic. The patient first turns over in the all-fours position and raises the trunk with his arms; the hands are then moved along the ground until the knees are reached; then with one hand upon a knee he lifts himself up, grasps the other knee, and gradually pushes himself into the erect posture, as it has been expressed, by climbing up his legs. The striking contrast between the feebleness of the child and the powerful-looking pseudo-hypertrophic muscles is very characteristic. The enlarged muscles may, however, be relatively very strong.

The course of the disease is slow, but progressive. Wasting proceeds and finally all traces of the enlarged condition of the muscles disappear. At this late period distortions and contractions are common.

The muscles of the shoulder-girdle are nearly always affected early in the disease, causing a symptom upon which Erb lays great stress. With the hands under the arms, when one endeavors to lift the patient, the shoulders are raised to the level of the ears, and one gets the impression as though the child were slipping through. These "loose shoulders" are very characteristic. The abnormal mobility of the shoulder-blades gives them a winged appearance, and makes the arms seem much longer than usual when they are stretched out.

The patients complain of no sensory symptoms. The atrophic muscles do not show the reaction of degeneration except in extremely rare instances.

Clinical Forms.—A number of different types have been described, depending upon the age at the onset, the muscles first affected, the occurrence of hypertrophy, the prominence of heredity, etc. But Erb has shown that there is no sharp division between these different forms, and classes them all under the name of *dystrophia muscularis progressiva*. For convenience of description he subdivides the disease into two large groups:

I. Those cases which occur in childhood.

II. The cases occurring in youth and adult life.

The first division is subdivided into (1) the hypertrophic and (2) the atrophic form.

Under the hypertrophic form, which is the pseudo-hypertrophic muscular paralysis of authors, he thinks it is useful to distinguish between the cases in which (*a*) the enlarged muscles have undergone lipomatosis—i. e., pseudo-hypertrophy—from those (*b*) in which there is a real hypertrophy.

The atrophic form also includes two subclasses: (*a*) Those cases in which the muscles of the face are involved early; this corresponds to the infantile form of Duchenne—the Landouzy-Dejerine type. (*b*) Those cases in which the face is not involved.

I. *Dystrophia muscularis progressiva infantum.*

1. Hypertrophic form.
   (*a*) With pseudo-hypertrophy.
   (*b*) With real hypertrophy.
2. Atrophic form.
   (*a*) With primary involvement of the face (infantile form of Duchenne).
   (*b*) Without involvement of the face.

II. *Dystrophia muscularis progressiva juvenum vel adultorum* (Erb's juvenile form).

Morbid Anatomy.—According to Erb, the disease consists in a change in the muscles themselves. At first the muscle-fibres hypertrophy, and become round; the nuclei increase, and the muscle-fibres may become fissured. At the same time there is a slight increase in the connective tissue. Sooner or later the muscle-fibres begin to atrophy, and the nuclei become greatly increased. Vacuoles and fissures appear, and the fibres finally become completely atrophic, the connective tissue becoming markedly increased. Fat may be deposited in the connective tissue to such an extent as to cause hypertrophic lipomatosis—pseudo-hypertrophy. The different stages of these changes may be found in a single muscle at the same time.

The nervous system has very generally been found to be without demonstrable lesions, but in certain cases changes in the cells of the ventral horn have been described.

Diagnosis.—The muscular dystrophies can usually be readily distinguished from the other forms of muscular atrophy.

(*a*) In the cerebral atrophy loss of power usually precedes the atrophy, which is either of a monoplegic or hemiplegic type.

(*b*) From progressive (central) muscular atrophy the distinctions are clearly marked. This form begins in the small muscles of the hand, a situation rarely if ever, affected by the dystrophies, which involve first those of the calves, the trunk, the face, or the shoulder-girdle. In the central atrophy the reaction of degeneration is present and fibrillary twitchings occur in both the atrophied and non-atrophied muscles. In many cases, in addition to the wasting in the arms, there is a spastic condition in the legs and increase in the reflexes. The central atrophies come on late in life; the dystrophies develop, as a rule, early. In the progressive muscular dys-

trophies heredity plays an important *rôle*, which in the central form is quite subsidiary. In the rare cases of early infantile spinal muscular atrophy occurring in families the symptoms are so characteristic of a central disease that the diagnosis presents no difficulty.

(*c*) In the neuritic muscular atrophies, whether due to lead or to trauma, the general characters and the mode of onset are distinctive. In the cases of multiple neuritis seen for the first time at a period when the wasting is marked there is often difficulty, but the absence of family history and the distribution are important features. Moreover, the paralysis is out of proportion to the atrophy. Sensory symptoms may be present, and in the cases in which the legs are chiefly involved there is usually the *steppage* gait so characteristic of peripheral neuritis.

(*d*) Progressive neural muscular atrophy. Here heredity is also a factor, and the disease usually begins in early life, but the distribution of atrophy and paralysis, which in this affection is at first confined to the periphery of the extremities, helps to distinguish it from the dystrophies; while the occurrence of sensory symptoms, fibrillary contractions, and the marked decrease in the electrical excitability usually make the distinction clear.

The outlook in the primary muscular dystrophies is bad. The wasting progresses uniformly, uninfluenced by treatment. Erb holds that by electricity and massage the progress is occasionally arrested. The general health should be carefully looked after, moderate exercise allowed, frictions with oil applied to the muscles, and when the patient becomes bedfast, as is inevitable sooner or later, care should be taken to prevent contractures in awkward positions.

The three forms of progressive muscular wasting—progressive (central) muscular atrophy, progressive neural muscular atrophy, and the muscular dystrophies—have been considered as distinct diseases, but certain recent writings make it probable that the distinction may not be so sharp as we believe. Certain cases occur which seem not to belong to any one of the forms but to stand between them. The changes in the muscles which were thought to be characteristic of the dystrophies have been found in the other forms. The central form occurs as a family disease in infancy, and the nervous system has been found diseased in the dystrophies.

The whole question is in a chaotic state, and it is at present better to keep to the old divisions. Even if it should turn out to be true, as Strümpell suggests, that all the forms depend upon a congenital tendency of the motor system to degenerate, they represent well-defined clinical types, into which the cases can, as a rule, be grouped without difficulty, while corresponding to each there is a fairly well-determined anatomical basis.

B. SYSTEM DISEASES OF THE UPPER MOTOR SEGMENT.

The question of an uncomplicated primary degeneration of the upper motor neurones has not been decided. Cases with a clinical picture corresponding to this lesion are not uncommon, and they may persist for a long time without change. Unfortunately the cases which have come to autopsy have shown various conditions. In only two or three has the disease been

so nearly confined to the pyramidal tract that they can be used as an argument for the independence of this condition. The cases of Minkowski, Dreschfeld, and Strümpell are not absolutely conclusive, as they are not quite pure, although they go far to prove that a degeneration in the pyramidal tract may be uncomplicated, at least for a long time. The same may be said for the group of cases described by Bernhardt and Strümpell under the name hereditary spastic spinal paralysis, in which the extensive systemic degeneration of the pyramidal tracts is combined with slight degeneration in other tracts of the cord.

1. Spastic Paralysis of Adults

(*Tabes dorsalis spasmodique; Primary Lateral Sclerosis*).

Definition.—A gradual loss of power with spasm of the muscles of the body, the lower extremities being first and most affected, unaccompanied by muscular atrophy, sensory disturbance, or other symptoms. The pathological anatomy is undetermined, but a systemic degeneration of the pyramidal tracts is assumed.

Symptoms.—The general *symptoms* of spastic paraplegia in adults are very distinctive. The patient complains of feeling tired, of stiffness in the legs, and perhaps of pains of a dull aching character in the back or in the calves. There may be no definite loss of power, even when the spastic condition is well established. In other instances there is definite weakness. The stiffness is felt most in the morning. In a well-developed case the gait is most characteristic. The legs are moved stiffly and with hesitation, the toes drag and catch against the ground, and, in extreme cases, when the ball of the foot rests upon the ground a distinct clonus develops. The legs are kept close together, the knees touch, and in certain cases the adductor spasm may cause cross-legged progression. On examination, the legs may at first appear tolerably supple, perhaps flexed and extended readily. In other cases the rigidity is marked, particularly when the limbs are extended. The spasm of the adductors of the thigh may be so extreme that the legs are separated with the greatest difficulty. In cases of this extreme rigidity the patient usually loses the power of walking. The nutrition is well maintained, the muscles may be hypertrophied. The reflexes are greatly increased. The slightest touch upon the patellar tendon produces an active knee-jerk. The rectus clonus and the ankle clonus are easily obtained. In some instances the slightest touch may throw the legs into violent clonic spasm, the condition to which Brown-Séquard gave the name of spinal epilepsy. The superficial reflexes are also increased. The arms may be unaffected for years, but occasionally they become weak and stiff at the same time as the legs. This was the case in a colored boy who was in my wards for several years. He presented a degree of general spastic rigidity that I have never seen equalled. The disease had begun after puberty, developed gradually, and remained quite stationary for more than a year before he left the wards. There were no other symptoms.

The course of the disease is progressively downward. Years may elapse before the patient is bedridden. Involvement of the sphincters, as a rule,

is late; occasionally, however, it is early. The sensory symptoms rarely progress, and the patients may retain their general nutrition and enjoy excellent health. Ocular symptoms are rare.

The *diagnosis*, so far as the clinical picture is concerned, is readily made, but it is often very difficult to determine accurately the nature of the underlying pathological condition. A history of syphilis is present in many of the cases. Cases which have run a fairly typical clinical course upon coming to autopsy have been found to have been due to very different conditions—transverse myelitis, multiple sclerosis, cerebral tumor, etc. General paralysis of the insane may begin with symptoms of spastic paraplegia, and Westphal believed that it was only in relation to this disease that a primary sclerosis of the pyramidal tracts ever occurred. In any case the diagnosis of primary systemic degeneration of the pyramidal tract is, to say the least, doubtful.

2. Spastic Paralysis of Infants—Spastic Diplegia—Birth Palsies

(*Paraplegia cerebralis spastica* (*Heine*); *Little's Disease*).

In this condition there is a paralysis with spasm of all extremities, dating from or shortly succeeding birth, more rarely following the fevers or an attack of convulsions. The legs are usually more involved than the arms; there is no wasting, no disturbance of sensation. The reflexes are increased. The mental condition is usually much disturbed. The patients are often imbeciles or idiots, helpless in mind and body. Ataxic and athetoid movements of the most exaggerated kind may occur.

While a limited number only of cases of infantile hemiplegia are congenital, on the other hand, in spastic diplegia and paraplegia a large proportion of the cases results from injury at birth. The arms may be so slightly affected as to make it difficult to determine whether it is a case of diplegia or paraplegia. The disease usually dates from birth, and a majority of the children are born in first labors or are forceps cases, and are at birth asphyxiated blue babies. Ross suggests that in feet presentations there may be laceration or tearing of the cerebro-spinal membranes. Premature birth is also given as a cause.

Morbid Anatomy.—The birth palsies which ultimately induce the spastic diplegias or paraplegias are most frequently the result of meningeal hæmorrhage. The importance of this condition has been shown by the studies of Litzmann and Sarah J. McNutt. The bleeding may come from the veins, or, as in one case which I saw with Hirst, from the longitudinal sinus. The hæmorrhage has in many cases been thickest over the motor areas, and it seems probable that the sclerosis found in these cases may result from compression by the blood-clot. In other instances the condition may be due to a fœtal meningo-encephalitis. In 16 autopsies collected in the literature, in which the patients died at ages varying from two to thirty, the anatomical condition was either a diffuse atrophy, which was most common, or porencephalus. From the fact that certain of the cases are born prematurely, before the pyramidal tracts are developed, it has been assumed by some that a non-development of these tracts is the cause of the

disease. This hypothesis has been urged by Marie, who limits the name spastic paraplegia to that group of the infantile cases in which there is no evidence of involvement of the brain—intellectual disturbances, epilepsy, etc., and it is in these cases that he believes the pyramidal tract has remained undeveloped.

Symptoms.—At first nothing abnormal may be noticed about the child. In some instances there have been early and frequent convulsions; then at the age when the child should begin to walk it is noticed that the limbs are not used readily, and on examination a stiffness of the legs and arms is found. Even at the age of two the child may not be able to sit up, and often the head is not well supported by the neck muscles. The rigidity, as a rule, is more marked in the legs, and there is adductor spasm. When supported on the feet, the child either rests on its toes and the inner surface of the feet, with the knees close together, or the legs may be crossed. The stiffness of the upper limbs varies. It may be scarcely noticeable or the rigidity may be as marked as in the legs. When the spastic condition affects the arms as well as the legs, we speak of the condition as diplegia; when the legs alone are involved, as paraplegia. There seems to be no sufficient reason for considering them separately. Constant irregular movements of the arms are not uncommon. The child has great difficulty in grasping an object. The spasm and weakness may be more evident on one side than the other. The mental condition is, as a rule, defective and convulsive seizures are common.

Associated with the spastic paralysis are two allied conditions of considerable interest, characterized by spasm and disordered movements. A child with spastic diplegia may present, in an unusual degree, irregular movements of the muscles. In attempting to grasp an object the fingers may be thrown out in a stiff, spasmodic, irregular manner, or there may be constant irregular movements of the shoulders, arms, and hands, with slight incoördination of the head. Cases of this description have been described as *chorea spastica*, and they may be difficult to separate from multiple sclerosis and from Friedreich's ataxia.

A still more remarkable condition is that of *bilateral athetosis*, in which there is a combination of spasm more or less marked with the most extraordinary bizarre movements of the muscles. The condition, as a rule, dates from infancy. The patient may not be able to walk. The head is turned from side to side; there are continual irregular movements of the face muscles, and the mouth is drawn and greatly distorted. The extremities are more or less rigid, particularly in extension. On the slightest attempt to move, often spontaneously, there are extraordinary movements of the arms and legs, particularly of the arms, somewhat like though much more exaggerated than athetosis. The patients are often unable to help themselves on account of these movements. The reflexes are increased. The mental condition is variable. The patient may be idiotic, but in 3 of the 6 cases which I have seen the patients were intelligent. Massalongo, who has carefully studied this condition, describes 3 cases in one family. I have collected 53 cases from the literature, 33 of which occurred in males and 20 in females.

3. Hereditary Spastic Paraplegia

(Hereditary Spastic Spinal Paralysis; Family form of Spastic Spinal Paralysis).

Much interest has been aroused in this type, cases of which have been described by Gee, Strümpell, Bernhardt, Latimer, Newmark, Erb, Tooth, Sachs, and others. Apparently we have to distinguish in this form two groups of cases. In one the disease develops in infancy or childhood, and the cases have all the characters of a *paraplegia spastica cerebralis*. In these cases, however, the symptoms pointing to disease of the brain, mental disturbances, epilepsy, etc., may be entirely wanting, and it was in relation to them that Erb made the suggestion that possibly too much stress had been laid upon the cerebral disease. He thought that a systemic degeneration of the lower part of the pyramidal tract accounted for the symptoms. The cases of amaurotic family idiocy described by Sachs, Peterson, Hirsch, and others do not belong here, although in them there is also a sclerosis of the pyramidal tract.

In the other group of cases, described by Bernhardt and Strümpell, the disease develops later, usually between twenty and thirty. The progress is very slow, extending over many years. At first there is no paralysis, only a spastic condition of the legs. The arms are affected later. Toward the end there may be a true paralysis, sensation may be affected, and the bladder may be slightly involved. In a fatal case of Strümpell's there was an extensive degeneration of the pyramidal tract and slight disease of the columns of Goll and of the direct cerebellar tract.

Amaurotic Family Idiocy.—A remarkable form of infantile paralysis has been described by Sachs, Peterson, and Hirsch. The symptoms as summarized by Sachs are: 1. Psychic disturbances that appear in early life (first or second year) and progress to total idiocy. 2. Paresis, and ultimately complete paralysis of the extremities, which may be either flaccid or spastic. 3. Increased, decreased, or normal tendon reflexes. 4. Partial, followed by total, blindness (macular changes, with subsequent atrophy of the optic nerve). 5. Marasmus and death, usually before the second year. 6. Distinct familial type. Occasional symptoms are nystagmus, strabismus, hyperacusis, or impairment of hearing. The pathological changes are primitive type of the cerebral convolutions, macrogyria, degenerative changes in the large pyramidal cells, absence of the tangential fibres, and decrease of the fibres of the white matter. The blood-vessels are normal. There is also degeneration of the pyramidal columns of the cord. Of 27 cases collected by Sachs, 17 occurred in six families; all in Jews.

4. Erb's Syphilitic Spinal Paralysis.

Erb has described a symptom group under the term syphilitic spinal paralysis, to which much attention has been given. The points upon which he lays stress are a very gradual onset with a development finally of the features of a spastic paresis; the tendon reflexes are greatly increased, but the muscular rigidity is slight in comparison with the exaggerated deep reflexes. There is rarely much pain, and the sensory disturbances are trivial,

but there may be paræsthesia and the girdle sensation. The bladder and rectum are usually involved, and there is sexual failure or impotence. And, lastly, improvement is not infrequent. A majority of instances of spastic paralysis of adults not the result of slow compression of the cord are associated with syphilis and belong to this group.

Erb thought the lesion to be a special form of transverse myelitis, but perhaps it should be classed with the system diseases, under the name toxic spastic spinal paralysis.

5. Secondary Spastic Paralysis.

Following any lesion of the pyramidal tract we may have spastic paralysis; thus, in a transverse lesion of the cord, whether the result of slow compression (as in caries), chronic myelitis, the pressure of tumor, chronic meningo-myelitis, or multiple sclerosis, degeneration takes place in the pyramidal tracts, below the point of disease. The legs soon become stiff and rigid, and the reflexes increase. Bastian has shown that in compression paraplegia if the transverse lesion is complete, the limbs may be flaccid, without increase in the reflexes—*paraplégie flasque* of the French. The condition of the patient in these secondary forms varies very much. In chronic myelitis or in multiple sclerosis he may be able to walk about, but with a characteristic spastic gait. In the compression myelitis, in fracture, or in caries, there may be complete loss of power with rigidity.

It may be difficult or even impossible to distinguish these cases from those of primary spastic paralysis. Reliance is to be placed upon the associated symptoms; when these are absent no definite diagnosis as to the cause of the spastic paralysis can be given.

6. Hysterical Spastic Paraplegia.

There is no spinal-cord disease which may be so accurately mimicked as spastic paraplegia. In the hysterical form there is wasting, the sensory symptoms are not marked, the loss of power is not complete, and there is not that extensor spasm so characteristic of organic disease. The reflexes are, as a rule, increased. The knee-jerk is present, and there may be a well-developed ankle clonus. Gowers calls attention to the fact that it is usually a spurious clonus, "due to a half-voluntary contraction in the calf muscles." A true clonus does occur, however, and there may be the greatest difficulty in determining whether or not the case is one of hysterical paraplegia. The hysterical contracture will be considered later.

C. SYSTEM DISEASES OF THE LOWER MOTOR SEGMENT.

1. Chronic Anterior Polio-myelitis

(Progressive Muscular Atrophy—Aran-Duchenne).

This disease has been considered as one of the types making up the progressive (central) muscular atrophies. In certain rare cases the process is confined to the lower motor segments. They, however, differ so little

clinically from many of the cases in which the pyramidal tracts are involved that it seems better to make no sharp distinction between them. The same may be said of chronic bulbar paralysis

2. Ophthalmoplegia.

This disease is at times due to a chronic degeneration of the nuclei of the motor nerves of the eyeballs, and so is a system disease of the lower motor segment. It is treated of in connection with the other ocular palsies for the sake of simplicity and because all ophthalmoplegias are not due to nuclear disease.

3. Acute Anterior Polio-myelitis

(Atrophic Spinal Paralysis; Infantile Paralysis).

This disease was formerly believed to be due to an acute inflammation of the cells of the ventral horns, depending upon a selective action of the poison for these cells, and would on this theory have properly been classed as a system disease of the lower motor neurones. Later observations indicate that the distribution of the inflammation depends upon the blood supply, and possibly that a thrombotic or an embolic process may act as the exciting cause of the inflammation. Just why this process should always act through the arteries supplying the ventral horns has not been explained. In any case the disease appears to be a focal inflammation, and not a system disease in the sense that the term is used in this work.

Clinically, the symptoms are confined to the motor system, and for this reason it is considered here and not with the focal lesions of the spinal cord, where our present views of its pathology would place it.

Definition.—An affection occurring most commonly within the first three years of life, characterized by fever, loss of power in certain muscles, and rapid atrophy.

Etiology.—The cause of the disease is unknown. It has been attributed to cold, to the irritation from dentition, or to overexertion. Since the days of Mephibosheth, parents have been inclined to attribute this form of paralysis to the carelessness of nurses in letting the children fall, but very rarely is the disease induced by traumatism, and in perhaps a majority of the cases the child is attacked while in full health. As Sinkler has pointed out, the cases are more common in the warm months. Boys are more liable to be affected than girls. Several instances of the occurrence of numerous cases together in epidemic form have been described. Medin reports from Stockholm an epidemic in which from the 9th of August to the 23d of September 29 cases came under observation. In two instances two children in the same family were attacked within a few days.

The most remarkable epidemic is that which occurred in the vicinity of Rutland, Vt., and which has been recorded by Caverly (New York Medical Record, 1894, ii). One hundred and nineteen cases occurred during the summer of 1894; 85 were under six years of age; 18 died.

Additional small outbreaks have been recorded of late years in New York and in London - Ontario.

Although most frequent in children, it develops occasionally in young adults, or even in middle-aged persons.

Morbid Anatomy.—The disease is oftenest seen in either the cervical or lumbar enlargements. In very early cases, such as those described by David Drummond and Charlewood Turner, the lesion has been that of an acute hæmorrhagic myelitis with degeneration and rapid destruction of the large ganglion cells. The condition may be strictly confined to the ventral cornua; in some instances there is slight meningeal involvement. The investigations of Goldscheider, Siemerling, and others have demonstrated the arterial origin of the disease, which is localized in the parts supplied by the ventral median branch of the ventral spinal artery. Occasionally the changes are found in the region of distribution of the ventral radicular arteries. Marie thinks that the initial process is embolism or thrombosis of the arteries of the ventral horns, the result of an acute infection. In cases in which the examination is not made for some months or years the changes are very characteristic. The ventral cornu in the affected region is greatly atrophied and the large motor cells are either entirely absent or only a few remain. The affected half of the cord may be considerably smaller than the other. The ventro-lateral column may show slight sclerotic changes, chiefly in the pyramidal tract. The corresponding ventral nerve roots are atrophied, and the muscles are wasted and gradually undergo a fatty and sclerotic change.

Symptoms.—In a majority of the cases, after slight indisposition and feverishness, the child is noticed to have lost the use of one limb. Convulsions at the outset are rare, not constant as in the acute cerebral palsies of children. Fever is usually present, the temperature rising to 101°, sometimes to 103°. Pain is often complained of in the early stages. This may be localized in the back or between the shoulders; any pressure on the paralyzed limbs may be painful, causing the patient to cry out when he is moved in bed. The paralysis is abrupt in its onset and, as a rule, is not progressive, but reaches its maximum in a very short time, even within twenty-four hours. It is rarely generalized. The suddenness of onset is remarkable and suggests a primary affection of the blood-vessels, a view which the hæmorrhagic character of the early lesion supports. The distribution of the paralysis is very variable. Its irregularity and lack of symmetry is quite characteristic of the disease. One or both arms may be affected, one arm and one leg, or both legs; or it may be a crossed paralysis, the right leg and the left arm. In the upper extremities the paralysis is rarely complete and groups of muscles may be affected. As Remak has pointed out, there is an upper-arm and a lower-arm type of palsy. The deltoid, the biceps, brachialis anticus, and supinator longus may be affected in the former, and in the latter the extensors or flexors of the fingers and wrists. This distribution is due to the fact that muscles acting functionally together are represented near each other in the spinal cord.

In the legs the tibialis anticus and extensor groups of muscles are more affected than the hamstrings and glutei. The muscles of the face are very rarely, the sphincters hardly ever involved. While the rule is for the paralysis to be abrupt and sudden, there are cases in which it comes

on slowly and takes from three to five days for its development. At first the affected limb looks natural, and as children between two and three are usually fat, very little change may be noticed for some time; but the atrophy proceeds rapidly, and the limb becomes flaccid and feels soft and flabby. Usually as early as the end of the first week the reaction of degeneration is present. The nerves are found to have lost their irritability. The muscles do not react to the induced current, but to the constant current they respond by a sluggish contraction, usually to a weaker current than is normal. The paralysis remains stationary for a time, and then there is gradual improvement. Complete recovery is rare, and, when the anatomical condition is considered, is scarcely to be expected. The large motor cells of the cornua, when thoroughly disintegrated, cannot be restored. In too many cases the improvement is only slight and permanent paralysis remains in certain groups. Sensation is unaffected; the skin reflexes are absent, and the deep reflexes in the affected muscles are usually lost.

When the paralysis persists the wasting is extreme, the growth of the bones of the affected limb is arrested, or at any rate retarded, and the joints may be very relaxed; as, for instance, when the deltoid is affected, the head of the humerus is no longer kept in contact with the glenoid cavity. In the later stages very serious deformities are produced by the contracture of the muscles.

Diagnosis.—The condition is only too evident in the majority of cases. There is a flaccid, flabby paralysis of one or more limbs which has set in abruptly. The rapid wasting, the lax state of the muscles, the electrical reactions, and the absence of reflexes distinguish it from the cerebral palsies. In multiple neuritis, a rare disease in childhood, the paralysis is bilaterally symmetrical, affects the muscles at the periphery of the limbs, and is combined with sensory symptoms. The pseudo-paresis of rickets is a condition to be carefully distinguished. In this the loss of power is in the legs, rapid atrophy is not present, certain movements are possible but painful. The general hyperæsthesia of the skin, the characteristic changes in the bones, and the diffuse sweats are present. Disease of the hip or knee may produce a pseudo-paralysis which can with care be readily distinguished.

Prognosis.—The outlook in any case for complete recovery is bad. The natural course of the disease must be borne in mind; the sudden onset, the rapid but not progressive loss of power, a stationary period, then marked improvement in certain muscle groups, and finally in many cases contractures and deformities. There is no other disease in which the physician is so often subject to unjust criticism, and the friends should be told at the outset that in the severe and extensive paralysis complete recovery should not be expected. The best to be hoped for is a gradual restoration of power in certain muscle groups. In estimating the probable grade of permanent paralysis, the electrical examination is of great value.

Treatment.—The treatment of acute infantile paralysis has a bright and a dark side. In a case of any extent complete recovery cannot be expected; on the other hand, it is remarkable how much improvement may

finally take place in a limb which is at first completely flaccid and helpless. The following treatment may be pursued: If seen in the febrile stage, a brisk laxative and a fever mixture may be given. The child should be in bed and the affected limb or limbs wrapped in cotton. As in the great majority of cases the damage is already done when the physician is called and the disease makes no further progress, the application of blisters and other forms of counter-irritation to the back is irrational and only cruel to the child.

The general nutrition should be carefully maintained by feeding the child well, and taking it out of doors every day. As soon as the child can bear friction the affected part should be carefully rubbed; at first once a day, subsequently morning and evening. Any intelligent mother can be taught systematically to rub, knead, and pinch the muscles, using either the bare hand or, better still, sweet oil or cod-liver oil. This is worth all the other measures advised in the disease, and should be systematically practised for months, or even, if necessary, a year or more. Electricity has a much more limited use, and cannot be compared with massage in maintaining the nutrition of the muscles. The faradic current should be applied to those muscles which respond. The essence of the treatment is in maintaining the nutrition of the muscles, so that in the gradual improvement which takes place in parts, at least, of the affected segments of the cord the motor impulses may have to deal with well-nourished, not atrophied muscle fibres.

Of medicines, in the early stage ergot and belladonna have been warmly recommended, but it is unlikely that they have the slightest influence. Later in the disease strychnia may be used with advantage in one or two minim doses of the liquor strychniæ, which, if it has no other effect, is a useful tonic.

The most distressing cases are those which come under the notice of the physician six, eight, or twelve months after the onset of the paralysis, when one leg or one arm or both legs are flaccid and have little or no motion. Can nothing be done? A careful electrical test should be made to ascertain which muscles respond. This may not be apparent at first, and several applications may be necessary before any contractility is noticed. With a few lessons an intelligent mother can be taught to use the electricity as well as to apply the massage. If in a case in which the paralysis has lasted for six or eight months no observable improvement takes place in the next six months with thorough and systematic treatment, little or no hope can be entertained of further change.

In the later stage care should be taken to prevent the deformities resulting from the contractions. Great benefit results from a carefully applied apparatus. The tendon transplantation introduced by Goldthwaite seems to be a distinct advantage in many cases. Eulenberg has recently reported a case (1898) in which the pes equinus was marked; he was able to afford notable relief by tendon implantation. Half of the tendo-Achilles and a part of the tendon of the soleus were implanted upon the tendons of the peroneus longus et brevis, the remaining half of the tendo-Achilles being divided. The transference of the functions from the flexors

to the pronators was satisfactorily accomplished, and the results were surprisingly beneficial.

4. Acute and Subacute Polio-myelitis in Adults.

An acute polio-myelitis in adults, the exact counterpart of the disease in children, is recognized. A majority, however, of the cases described under this heading have been multiple neuritis; but the suddenness of onset, the rapid wasting, and the marked reaction of degeneration are thought by some to be distinguishing features. Multiple neuritis may, however, set in with rapidity; there may be great wasting and the reaction of degeneration is sometimes present. The time element alone may determine the true nature. Recovery in a case of extensive multiple paralysis from polio-myelitis will certainly be with loss of power in certain groups of muscles; whereas, in multiple neuritis the recovery, while slow, may be perfect.

The subacute form, the *paralysie générale spinale antérieure subaiguë* of Duchenne, is in all probability a peripheral palsy. The paralysis usually begins in the legs with atrophy of the muscles, then the arms are involved, but not the face. Sensation is, as a rule, not involved.

5. Acute Ascending (Landry's) Paralysis.

Definition.—An advancing paralysis, beginning in the legs, rapidly extending to the trunk and arms, and finally, in many cases, involving the muscles of respiration. It presents a remarkable similarity in its symptoms to certain cases of polyneuritis, with which it is now grouped by many writers.

Etiology and Pathology.—This disease occurs most commonly in males between the twentieth and thirtieth years. It has sometimes followed the specific fevers. An elaborate study of 93 cases collected from the literature has been made by James Ross, who concludes that in etiology, symptoms, course, and termination it conforms to a peripheral neuritis. Neuwerk and Barth have reached a similar conclusion. In their case an interstitial neuritis was found in the nerve roots, but the peripheral nerves were normal. Spiller found in a rapidly fatal case destructive changes in the peripheral nerves and corresponding alterations in the cell bodies of the ventral horns. He suggests that the toxic agent acts on the lower motor neurones as a whole, and that possibly the reason why no lesions were found in some of the cases is that the more delicate histological methods were not used. We may regard the disease, then, as an acute poisoning of the lower motor neurones.

Symptoms.—Weakness of the legs, gradually progressing, often with tolerable rapidity, is the first symptom. In some cases within a few hours the paralysis of the legs becomes complete. The muscles of the trunk are next affected, and within a few days, or even less in more acute cases, the arms are also involved. The neck muscles are next attacked, and finally the muscles of respiration, deglutition, and articulation. The reflexes are

Some 50 cases are on record and have been analy
and by Edson Harvey Campbell & Edward Brammwell (Brain 1901)
The disease is uncommon & among persons are chiefly affected. The
muscles innervated by the bulb are first attacked - those of the eyes the face
masticatory & of the neck. all the voluntary muscles may become
involved often next the lower & recovered. In severe cases paralysis
may persist. ~~The affected muscles may show~~ The myasthenic reaction
of Jolly is the rapid exhaustion of the muscles by faradism - not by the
galvanism. There are ~~marked~~ marked reactions & fluctuations in the
severity of the symptoms. The affected muscles in a few cases have
atrophied. In seven cases autopsies in only six was anything abnormal
found (Ch & B), and the significance of these changes is doubtful.
The diagnosis is easy from the ptosis, the facial expression & the nasal
speech the rapid fatigue of the muscles, the myasthenic reaction, the absence
of atrophy Tremors &c & the remarkable variations in the intensity of the
symptoms. Of the 60 cases 23 ended fatally. The patient may live many
years recovery may take place. Rest, strychnia & iron are useful
Marriage. Maternal calomel of 5 grains of potassium & mixing may be
tried

lost, but the muscles neither waste nor show electrical changes. The sensory symptoms are variable; in some cases tingling, numbness, and hyperæsthesia have been present. In the more characteristic cases sensation is intact and the sphincters are uninvolved. Enlargement of the spleen has been noticed in several cases. The course of the disease is variable. It may prove fatal in less than two days. Other cases persist for a week or for two weeks. In some instances recovery has occurred, but in a large proportion of the cases the disease is fatal.

The *diagnosis* is difficult, particularly from certain forms of multiple neuritis, and if we include in Landry's paralysis the cases in which sensation is involved, distinction between the two affections is impossible. We apparently have to recognize the existence of a rapidly advancing motor paralysis without involvement of the sphincters, without wasting or electrical changes in the muscles, without trophic lesions, and without fever—features sufficient to distinguish it from either the acute central myelitis or the polio-myelitis anterior. It is doubtful, however, whether these characters always suffice to enable us to differentiate the cases of multiple neuritis.

6. Asthenic (Bulbar) Paralysis

(*Myasthenia gravis pseudo-paralytica*; *Erb-Goldflam's Symptom-complex*).

During the last few years much attention has been given to this remarkable affection, of which a number of cases have been reported. The chief characteristics are the rapidity with which the muscles become exhausted, the great variability of the symptoms from day to day, the occurrence of remissions and relapses, the sudden attacks of paralysis of respiration and deglutition, and the absence of muscular atrophy, the reaction of degeneration and sensory symptoms. The onset is usually acute or subacute, chiefly in young persons. The external eye muscles, the muscles of mastication, the facial muscles, the muscles of deglutition, and certain spinal muscles may be quickly involved. Any repeated efforts with the affected muscles causes them to become completely exhausted and paralyzed for the time being. They recover their power after a rest. In certain cases there is a true paresis, which persists. After repeated stimulation by electricity the muscles may become exhausted and cease to respond (myasthenic reaction, Wolly). The affection may prove fatal, and as no well-defined anatomical lesions have been discovered, a dynamic change in the lower motor neurones has been assumed to explain the condition.

IV. COMBINED SYSTEM DISEASES.

When the disease is not confined within the limits of either the afferent or efferent systems, but affects both, it is known as a *combined system disease*. Some authors contend that the diseases usually classed under this head are not really system diseases, but are diffuse processes. This is the view taken by Leyden and Goldscheider, who limit the term system disease to locomotor ataxia and progressive muscular atrophy.

In certain cases of locomotor ataxia which have run a fairly typical course there may be found after death, besides the anatomical picture corresponding to this disease, a moderate degeneration of the pyramidal tracts and of the ventral horns. In progressive muscular atrophy, on the other hand, there may be degeneration in the dorsal column. During life these secondary involvements of other systems, as they may be termed, may or may not be accompanied by demonstrable symptoms, and when such do occur they make their appearance late in the disease.

There is another group of cases in which from the very first the symptoms point to an involvement of both the afferent and efferent systems, and it is to these that the term primary combined system disease is usually limited.

1. Ataxic Paraplegia.

This name is applied by Gowers to a disease characterized clinically by a combination of ataxia and spastic paraplegia, and anatomically by involvement of the dorsal and lateral columns.

The disease is most common in middle-aged males. Exposure to cold and traumatism have been occasional antecedents. In striking contrast to ordinary tabes a history of syphilis is rarely to be obtained.

The anatomical features are a sclerosis of the dorsal columns, which is not more marked in the lumbar region and not specially localized in the root zone of the cuneate fasciculi. The involvement of the lateral columns is diffuse, not always limited to the pyramidal tracts, and there may be an annular sclerosis. Marie believes that in many cases the distribution of the sclerosis is due to the arterial supply and not to a true systemic degeneration, the vessels involved being branches of the dorsal spinal artery.

The *symptoms* are well defined. The patient complains of a tired feeling in the legs, not often of actual pain. The sensory symptoms of true tabes are absent. An unsteadiness in the gait gradually develops with progressive weakness. The reflexes are increased from the outset, and there may be well-developed ankle clonus. Rigidity of the legs slowly comes on, but it is rarely so marked as in the uncomplicated cases of lateral sclerosis. From the start incoördination is a well-characterized feature, and the difficulty of walking in the dark or swaying when the eyes are closed may, as in true tabes, be the first symptom to attract attention. In walking the patient uses a stick, keeps the eyes fixed on the ground, the legs far apart, but the stamping gait, with elevation and sudden descent of the feet, is not often seen. The incoördination may extend to the arms. Sensory symptoms are rare, but Gowers calls attention to a dull, aching pain in the sacral region. The sphincters usually become involved. Eye symptoms are rare. Late in the disease mental symptoms may develop, similar to those of general paresis.

In well-marked cases the *diagnosis* is easy. The combination of marked incoördination with retention of the reflexes and more or less spasm are characteristic features. The absence of ocular and sensory symptoms is an important point.

2. Primary Combined Sclerosis (Putnam).

In addition to the ataxic paraplegia just mentioned, here may be considered certain cases which are characterized anatomically by a relatively chronic sclerosis of the dorsal columns, of the lateral columns, chiefly the pyramidal tract, and also of the cerebellar tract. With these are usually associated more acute changes in adjoining areas, either diffuse or systemic, some grade of degeneration in the gray matter, and involvement of the nerve roots. This form has been studied by J. J. Putnam and Dana. The cases are usually in women—7 out of 19 collected by Dana; the ages, from forty-five to sixty-four. The disease runs a rather rapid course. Neuropathic inheritance is present in some instances. Putnam thinks that possibly both lead and arsenic play a part in the etiology.

The *symptoms* are both sensory and motor. The onset is usually with numbness in the extremities, progressive loss of strength, and emaciation. Paraplegia gradually develops, before which there have been, as a rule, spastic symptoms with exaggerated knee-jerk. The arms are affected less than the legs. Mental symptoms suggestive of dementia paralytica may develop toward the close.

The *diagnosis* of this mixed sclerosis rests upon the combination of sensory and motor symptoms with the presence of exaggerated reflexes. As stated, the sensory features consist chiefly of paræsthesia, and there may be difficulty in distinguishing the condition from multiple neuritis. The frequency of the disease in more or less enfeebled or anæmic women past middle life is also an important feature.

3. Hereditary Ataxia (*Friedreich's Ataxia*).

In 1861 Friedreich reported 6 cases of a form of hereditary ataxia, and the affection has usually gone by his name. Unfortunately, *paramyoclonus multiplex* is also called Friedreich's disease; so it is best, if his name is used in connection with this affection, to term it Friedreich's ataxia. It is a very different disease in many respects from ordinary tabes. It may or may not be hereditary. It is really a family disease, several brothers and sisters being, as a rule, affected. The 143 cases analyzed by Griffith occurred in 71 unrelated families. In his series inheritance of the disease itself occurred in only 33 cases. Various influences in the parents have been noted; alcoholism in only 7 cases. Syphilis has rarely been present. Of the 143 cases, 86 were males and 57 females. The disease sets in early in life, and in Griffith's series 15 occurred before the age of two years, 39 before the sixth year, 45 between the sixth and tenth years, 20 between the eleventh and fifteenth years, 18 between the sixteenth and twentieth years, and 5 between the twentieth and twenty-fifth years.

The *morbid anatomy* shows an extensive sclerosis of the dorsal and lateral columns of the spinal cord. The periphery, and the cerebellar tracts are usually involved. The observations of Dejerine and Letulle are of special interest, since they seem to indicate that the change in this disease is

a neurogliar (ectodermal) sclerosis, differing entirely from the ordinary spinal sclerosis. According to this view, Friedreich's disease is a gliosis of the dorsal columns due to developmental errors; but the question is still unsettled.

Symptoms.—The ataxia differs somewhat from the ordinary form. The incoördination begins in the legs, but the gait is peculiar. It is swaying, irregular, and more like that of a drunken man. There is not the characteristic stamping gait of the true tabes. Romberg's symptom may or may not be present. The ataxia of the arms occurs early and is very marked; the movements are almost choreiform, irregular, and somewhat swaying. In making any voluntary movement the action is overdone, the prehension is claw-like, and the fingers may be spread or overextended just before grasping an object. The hand frequently moves about an object for a moment and then suddenly pounces upon it. There are irregular, swaying movements, some of which are choreiform, of the head and shoulders. There is present in many cases what is known as static ataxia, that is to say, ataxia of quiet action. It occurs when the body is held erect or when a limb is extended—irregular, oscillating movements of the head and body or of the extended limb.

Sensory symptoms are not usually present. The deep reflexes are lost early in the disease, and, next to the ataxia, this is the most constant and important symptom (Strümpell). The skin reflexes are usually normal, and the pupillary reflex to light is practically never affected.

Nystagmus is a characteristic symptom. Atrophy of the optic nerve rarely occurs. A striking feature is early deformity of the feet. There is talipes equinus, and the patient walks on the outer edge of the feet. The big toe is flexed dorsally on the first phalanx. Lateral curvature of the spine is very common.

Trophic lesions are rare. As the disease advances paralysis comes on and may ultimately be complete. Some of the patients never walk.

Disturbance of speech is common. It is usually slow and scanning; the expression is often dull; the mental power is, as a rule, maintained, but late in the disease becomes impaired.

The *diagnosis* of the disease is not difficult when several members of a family are affected. The onset in childhood, the curious form of incoördination, the loss of knee-kicks, the early talipes equinus, the position of the great toe, the scoliosis, the nystagmus, and scanning speech make up an unmistakable picture. The disease is often confounded with chorea, with the ordinary form of which it has nothing in common. With hereditary chorea it has certain similarities, but usually this disease does not set in until after the thirtieth year.

The affection lasts for many years and is incurable. Care should be taken to prevent contractures.

Cerebellar Type.—There is a form of hereditary ataxia, described by Marie as *cerebellar heredo-ataxia*, which starts later in life, after the age of twenty, with disability in the legs, but the gait is less ataxic than "groggy." The knee-jerks are retained, and a spastic condition of the legs ultimately develops. There is no scoliosis, nor does club-foot develop. Sanger Brown's

cases, 25 in one family, and J. H. Neff's, 13, appear to belong to this type. The cerebellum has been found atrophied in 2 cases.

4. Progressive Interstitial Hypertrophic Neuritis of Infants.

Under this imposing title Dejerine and Sottas described a rare and interesting affection. It is a family disease, and begins in early life. The symptoms are those typical of locomotor ataxia, to which is added progressive muscular atrophy, with involvement of the face and a hypertrophy and hardening of the peripheral nerves. As the name indicates, it is an interstitial hypertrophic neuritis with secondary involvement of the dorsal columns of the cord. This disease has been associated with progressive neural muscular atrophy, but Dejerine has shown that it is quite distinct.

5. Toxic Combined Sclerosis.

Certain poisons cause changes in the lateral and dorsal columns of the cord that resemble those of the combined system diseases. They have been demonstrated in pellagra and in ergotism, and have already been described. In pernicious anæmia and many chronic wasting disease these scleroses occur, and are believed to be due to the action of poisons produced within the system.

III. DIFFUSE DISEASES OF THE NERVOUS SYSTEM.

I. AFFECTIONS OF THE MENINGES.

Diseases of the Dura Mater (*Pachymeningitis*).

Pachymeningitis Externa.—*Cerebral.*—Hæmorrhage often occurs as a result of fracture. Inflammation of the external layer of the dura is rare. Caries of the bone, either extension from middle-ear disease or due to syphilis, is the principal cause. In the syphilitic cases there may be a great thickening of the inner table and a large collection of pus between the dura and the bone.

Occasionally the pus is infiltrated between the two layers of the dura mater or may extend through and cause a dura-arachnitis.

The symptoms of external pachymeningitis are indefinite. In the syphilitic cases there may be a small sinus communicating with the exterior. Compression symptoms may occur with or without paralysis.

Spinal.—An acute form may occur in syphilitic affections of the bones, in tumors, and in aneurism. The symptoms are those of a compression of the cord. A chronic form is much more common, and is a constant accompaniment of tuberculous caries of the spine. The internal surface of the dura may be smooth, while the external is rough and covered with caseous masses. The entire dura may be surrounded or the process may be confined to the ventral surface.

Pachymeningitis Interna.—This occurs in three forms: (1) Pseudo-membranous, (2) purulent, and (3) hæmorrhagic. The first two are unimportant. Pseudo-membranous inflammation of the lining membrane of the dura is not usually recognized, but a most characteristic example of it came under my observation as a secondary process in pneumonia. Purulent pachymeningitis may follow an injury, but is more commonly the result of extension from inflammation of the pia. It is remarkable how rarely pus is found between the dura and arachnoid membranes.

Hæmorrhagic Pachymeningitis (*Hæmatoma of the Dura Mater*).

Cerebral Form.—This remarkable condition, first described by Virchow, is very rare in general medical practice. During ten years no instance of it came under my observation at the Montreal General Hospital. On the other hand, in the post-mortem room of the Philadelphia Hospital, which received material from a large almshouse and asylum, the cases were not uncommon, and within three months I saw four characteristic examples, three of which came from the medical wards. The frequency of the condition in asylum work may be gathered from the fact that in 1,185 post mortems at the Government Hospital for the Insane, Washington, to June 30, 1897, there were 197 cases with "a true neo-membrane of internal pachymeningitis" (Blackburn). Of these cases, 45 were chronic dementia, 37 were general paresis, 30 senile dementia, 28 chronic mania, 28 chronic melancholia, 22 chronic epileptic insanity, 6 acute mania, and 1 case imbecility. Forty-two of the cases were in persons over seventy years of age.

It has also been found in profound anæmia and other diseases of the blood and of the blood-vessels, and is said to have followed certain of the acute fevers. Herter has called attention to the not infrequent occurrence of the lesion in badly nourished, cachectic children.

The morbid anatomy is interesting. Virchow's view that the delicate vascular membrane precedes the hæmorrhage is undoubtedly correct. Practically we see one of three conditions in these cases: (*a*) Subdural vascular membranes, often of extreme delicacy, formed by the penetration of blood-vessels and granulation tissue into an inflammatory exudate (so-called "organization" of an inflammatory exudate); (*b*) simple subdural hæmorrhage; (*c*) a combination of the two, vascular membrane and blood-clot. Certainly the vascular membrane may exist without a trace of hæmorrhage—simply a fibrous sheet of varying thickness, permeated with large vessels, which may form beautiful arborescent tufts. On the other hand, there are instances in which the subdural hæmorrhage is found alone, but it is possible that in some of these at least the hæmorrhage may have destroyed all trace of the vascular membrane. In some cases a series of laminated clots are found, forming a layer from 3 to 5 mm. in thickness. Cysts may occur within this membrane. The source of the hæmorrhage is probably the dural vessels. Huguenin and others hold that the bleeding comes from the vessels of the pia mater, but certainly in the early stage of the condition there is no evidence of this; on the other hand, the highly vascular subdural membrane may be seen covered with the thinnest possible sheeting

of clot, which has evidently come from the dura. The subdural hæmorrhage is usually associated with atrophy of the convolutions, and it is held that this is one reason why it is so common in the insane, especially in dementia paralytica and dementia senilis; but there must be some other factor than atrophy, or we should meet with it in phthisis and various cachectic conditions in which the cerebral wasting is as common and almost as marked as in cases of insanity.

The symptoms are indefinite, or there may be none at all, especially when the hæmorrhages are small or have occurred very gradually, and the diagnosis cannot be made with certainty. Headache has been a prominent symptom in some cases, and when the condition exists on one side there may be hemiplegia. The most helpful symptoms for diagnosis, indicating that the hæmorrhage in an apoplectic attack is meningeal, are (1) those referable to increased intracerebral pressure (slowing and irregularity of the pulse, vomiting, coma, contracted pupils reacting to light slowly or not at all) and (2) paresis and paralysis, gradually increasing in extent, accompanied by symptoms which point to a *cortical* origin. Extensive bilateral disease may, however, exist without any symptoms whatever.

Spinal Form.—The spinal *pachymeningitis interna*, described by Charcot and Joffroy, involves chiefly the cervical region (*P. cervicalis hypertrophica*). The interspace between the cord and the dura is occupied by a firm, concentrically arranged, fibrinous growth, which is seen to have developed within, not outside of, the dura mater. It is a condition anatomically identical with the hæmorrhagic pachymeningitis interna of the brain. The cord is usually compressed; the central canal may be dilated—hydromyelus—and there are secondary degenerations. The nerve roots are involved in the growth and are damaged and compressed. The extent is variable. It may be limited to one segment, but more commonly involves a considerable portion of the cervical enlargement. The disease is chronic, and in some cases presents a characteristic group of symptoms. There are intense neuralgic pains in the course of the nerves whose roots are involved. They are chiefly in the arms and in the cervical region, and vary greatly in intensity. There may be hyperæsthesia with numbness and tingling; atrophic changes may develop, and there may be areas of anæsthesia. Gradually motor disturbances appear; the arms become weak and the muscles atrophied, particularly in certain groups, as the flexors of the hand. The extensors, on the other hand, remain intact, so that the condition of claw-hand is gradually produced. The grade of the atrophy depends much upon the extent of involvement of the cervical nerve roots, and in many cases the atrophy of the muscles of the shoulders and arms becomes extreme. The condition is one of cervical paraplegia, with contractures, flexion of the wrist, and typical *main en griffe*. Usually before the arms are greatly atrophied there are the symptoms of what the French writers term the second stage—namely, involvement of the lower extremities and the gradual production of a spastic paraplegia, which may develop several months after the onset of the disease, and is due to secondary changes in the cord.

The disease runs a chronic course, lasting, perhaps, two or more years.

In a few instances, in which symptoms pointed definitely to this condition, recovery has taken place. The disease is to be distinguished from amyotrophic lateral sclerosis, syringomyelia, and tumors. From the first it is separated by the marked severity of the initial pains in the neck and arms; from the second by the absence of the sensory changes characteristic of syringomyelia. From certain tumors it is very difficult to distinguish; in fact, the fibrinous layers form a tumor around the cord.

The condition known as *hæmatoma* of the dura mater may occur at any part of the cord, or, in its slow, progressive form—pachymeningitis hæmorrhagica interna—may be limited to the cervical region and produce the symptoms just mentioned. It is sometimes extensive, and may coexist with a similar condition of the cerebral dura. Cysts may occur filled with hæmorrhagic contents.

Diseases of the Pia Mater (*Acute Cerebro-spinal Leptomeningitis*).

Etiology.—Under cerebro-spinal fever and tuberculosis the two most important forms of meningitis have been described. Other conditions with which meningitis is associated are: (1) *The acute fevers*, more particularly pneumonia, erysipelas, and septicæmia; less frequently small-pox, typhoid fever, scarlet fever, measles, etc. (2) *Injury or disease of the bones of the skull.* In this group by far the most frequent cause is necrosis of the petrous portion of the temporal bone in chronic otitis. (3) *Extension from disease of the nose.* Meningitis has followed perforation of the skull in sounding the frontal sinuses, suppurative disease of these sinuses, and necroses of the cribriform plate. As mentioned under cerebro-spinal fever, the infection is thought to be possible through the nose. (4) As a *terminal infection* in chronic nephritis, arterio-sclerosis, heart-disease, gout, and the wasting diseases of children. Bacteriologically, we may recognize four great groups of meningitides—the form due to the meningococcus (diplococus intracellularis), the pneumococcus meningitis, the form due to the tubercle bacillus, and the streptococcus meningitis. The gonococcus, the typhoid bacillus, the colon bacillus, and staphylococci also cause meningitis, but a great majority of all the cases are due to the four first-mentioned micro-organisms. I have already spoken of the pneumococcus meningitis, which not only occurs in connection with pneumonia, but as an independent infection. A majority of all the cases of so-called sporadic meningitis are probably caused by it.

The streptococcus meningitis is the usual form in the cases due to trauma, to otitis media, and in septic processes. In ulcerative endocarditis it is not uncommon; it occurred in 25 of 209 collected cases.

The terminal meningitides are caused by the streptococci, sometimes by staphylococci.

Morbid Anatomy.—The basal or cortical meninges may be chiefly attacked. The degree of involvement of the spinal meninges varies. In the form associated with pneumonia and ulcerative endocarditis the disease is bilateral and usually limited to the cortex. In extension from disease of the ear it is often unilateral and may be accompanied with abscess or with

Margaret Mercury

thrombosis of the sinuses. In the non-tuberculous form in children, in the meningitis of chronic Bright's disease, and in cachectic conditions the base is usually involved. In the cases secondary to pneumonia the effusion beneath the arachnoid may be very thick and purulent, completely hiding the convolutions. The ventricles also may be involved, though in these simple forms they rarely present the distention and softening which is so frequent in the tuberculous meningitis. For a more detailed description the student is referred to the sections on cerebro-spinal fever and tuberculous meningitis.

Symptoms.—The clinical features of meningitis have already been described at length in the diseases just referred to, and I shall here give a general summary. I have already, on several occasions, called attention to the fact that cortical meningitis is not to be recognized by any symptoms or set of symptoms from a condition which may be produced by the poison of many of the specific fevers. In the cases of so-called cerebral pneumonia, unless the base is involved and the nerves affected, the disease is unrecognizable, since identical symptoms may be produced by intense engorgement of the meninges. In typhoid fever, in which meningitis is very rare, the twitchings, spasms, and retractions of the neck are almost invariably associated with cerebro-spinal congestion, not with meningitis. Actual meningitis does, however, occur in typhoid fever, and, as Ohlmacher's cases show, the typhoid bacilli may be present in the exudate.

A knowledge of the etiology gives a very important clew. Thus, in middle-ear disease the development of high fever, delirium, vomiting, convulsions, and retraction of the head and neck would be extremely suggestive of meningitis or abscess. Headache, which may be severe and continuous, is the most common symptom. While the patient remains conscious this is usually the chief complaint, and even when semicomatose he may continue to groan and to place his hand on his head. In the fevers, particularly in pneumonia, there may be no complaint of headache. Delirium is frequently early, and is most marked when the fever is high. Convulsions are less common in simple than in tuberculous meningitis. They were not present in a single instance in the cases which I have seen in pneumonia, ulcerative endocarditis, or septicæmia. In the simple meningitis of children they may occur. Epileptiform attacks which come and go are highly characteristic of direct irritation of the cortex. Rigidity and spasm or twitchings of the muscles are more common. Stiffness and retraction of the muscles of the neck are important symptoms; but they are by no means constant, and are most frequent when the inflammation is extensive on the meninges of the cervical cord. There may be trismus, gritting of the teeth, or spastic contraction of the abdominal muscles. Vomiting is a common symptom in the early stages, particularly in basilar meningitis. Constipation is usually present. In the late stages the urine and fæces may be passed involuntarily. Optic neuritis is rare in the meningitis of the cortex, but is not uncommon when the base is involved. Leube lays stress on the hyperæsthesia of the skin and muscles, especially of the muscles of the neck and calves.

Important symptoms are due to lesions of the nerves at the base. Stra-

bismus or ptosis may occur. The facial nerve may be involved, producing slight paralysis, or there may be damage to the fifth nerve, producing anæsthesia and, if the Gasserian ganglion is affected, trophic changes in the cornea. The pupils are at first contracted, subsequently dilated, and perhaps unequal. The reflexes in the extremities are often accentuated at the beginning of the disease; later they are diminished or entirely abolished. Herpes is common, particularly in the epidemic form.

Fever is present, moderate in grade, rarely rising above 103°. In the non-tuberculous leptomeningitis of debilitated children and in Bright's disease there may be little or no fever. The pulse may be increased in frequency at first, though this is unusual. One of the striking features of the disease is the slowness of the pulse in relation to the temperature, even in the early stages. Subsequently it may be irregular and still slower. The very rapid emaciation which often occurs is doubtless to be referred to a disturbance of the cerebral influence upon metabolism. The spinal meninges are so often affected simultaneously that lumbar puncture is exceedingly valuable for diagnosis. Not only does this frequently prove indisputably the existence of an acute meningitis, but the bacteriological examination may decide as to the etiological factor, and thus yield a more rational basis for treatment.

Treatment.—There are no remedies which in any way control the course of acute meningitis. An ice-bag should be applied to the head and, if the subject is young and full-blooded, general or local depletion may be practised. Absolute rest and quiet should be enjoined. When disease of the ear is present, a surgeon should be early called in consultation, and if there are symptoms of meningo-encephalitis which can in any way be localized trephining should be practised. An occasional saline purge will do more to relieve the congestion than blisters and local depletion. I have no belief whatever in the efficacy of counter-irritation to the back of the neck, and to apply a blister to a patient suffering with agonizing headache in meningitis is needlessly to add to the suffering. If counter-irritation is deemed essential, the thermo-cautery, lightly applied, is more satisfactory. Large doses of the perchloride of iron, iodide of potassium, and mercury are recommended by some authors.

The application of an ice-cap, attention to the bowels and stomach, and keeping the fever within moderate limits by sponging, are the necessary measures in a disease recognized as almost invariably fatal, and in which the cases of recovery are extremely doubtful. Quincke's lumbar puncture (see page 107) has been used as a therapeutic measure with success by Fürbringer; 60 cc. of cloudy fluid were removed, in which tubercle bacilli were found. The headache and other cerebral symptoms disappeared, and the patient, a man of twenty, recovered. Wallis Ord and Waterhouse report a case of recovery, in a child of five years, after trephining and drainage.

Posterior Meningitis of Infants (*Non-tuberculous Leptomeningitis Infantum*).

This form has been specially studied by Gee and Barlow, and has been called occlusive meningitis. Gee called it *cervical opisthotonos* of infants, from the most prominent feature of the disease. A careful study has been made of 11 cases by J. W. Carr. In all cases there was well-marked distention of the lateral and third ventricles, generally of the fourth also, with "effusion of lymph, thickening of the pia-arachnoid, and matting of the parts over the posterior and central area of the base of the brain from the lower end of the medulla to the optic commissure." The disease is most common in infants under one year. In only 3 cases a few flakes of lymph were found, and neither the choroid plexuses nor the ependema showed naked-eye appearances of inflammation. Head retraction appeared early and was persistent throughout, being absent in only one case. It is usually much more marked than in tuberculous meningitis. At a comparatively early stage, even weeks before death, the infants pass into stupor or complete coma. This form is sometimes met with in older children.

Chronic Leptomeningitis.—This is rarely seen apart from syphilis or tuberculosis, in which the meningitis is associated with the growth of the granulomata in the meninges and about the vessels. The symptoms in such cases are extremely variable, depending entirely upon the situation of the growth. They may closely resemble those of tumor and be associated with localized convulsions. The epidemic meningitis may run a very chronic course. The leptomeningitis infantum may be chronic. In the cases reported by Gee and Barlow the duration in some instances extended even to a year and a half. Quincke's *meningitis serosa* is considered with hydrocephalus.

II. SCLEROSES OF THE BRAIN.

General Remarks.—The connective tissue of the central nervous system is of two kinds—one, the neuroglia, special and peculiar, derived from the ectoderm, with distinct morphological and chemical characters; the other, in the meninges and accompanying the blood-vessels, derived from the mesoderm, identical with the ordinary collagenous fibrous tissue of the body. Both play important parts in indurative processes in the brain and cord. A convenient division of the cerebro-spinal scleroses is into degenerative, inflammatory, and developmental forms.

The *degenerative scleroses* comprise the largest and most important subdivision, in which provisionally the following groups may be made: (*a*) The common secondary degeneration which follows when nerve-fibres are cut off from their trophic centres (the severance of portions of neurones from the main portions containing the nuclei); (*b*) toxic forms, among which may be placed the scleroses from lead and ergot, and, most important of all, the sclerosis of the dorsal columns, due in such a large proportion of cases to the virus of syphilis. Other unknown toxic agents may possibly induce

degeneration of the nerve-fibres in certain tracts. The systemic paths in the cord differ apparently in their susceptibility and the dorsal columns appear most prone to undergo this change; (*c*) the sclerosis associated with change in the smaller arteries and capillaries, which is met with as a senile process in the convolutions. In all probability some of the forms of insular sclerosis are due to primary alterations in the blood-vessels; but it is not yet settled whether the lesion in these cases is a primary degeneration of the nerve cells and fibres to which the sclerosis is secondary, or whether the essential factor is an alteration in nutrition caused by lesions of the capillaries and smaller arteries.

The *inflammatory scleroses* embrace a less important and less extensive group, comprising secondary forms which develop in consequence of irritative inflammation about tumors, foreign bodies, hæmorrhages, and abscess. Histologically these are chiefly mesodermic (vascular) scleroses, which arise from the connective tissue about the blood-vessels. Possibly a similar change may follow the primary, acute encephalitis, which Strümpell holds is the initial lesion in the cortical sclerosis which is so commonly found post mortem in infantile hemiplegia.

The *developmental scleroses* are believed to be of a purely neurogliar character, and embrace the new growth about the central canal in syringomyelia and, according to recent French writers, the sclerosis of the dorsal columns in Friedreich's ataxia. It is stated that histologically this form is different from the ordinary variety. It may be, too, that the diffuse cortical sclerosis met with as a congenital condition without thickening of the meninges belongs to this type. It is not improbable that many forms of scleroses are of a mixed character, in which both the ectodermic glia and mesodermic connective tissue are involved.

Anatomically we meet with the following varieties:

(1) **Miliary sclerosis** is a term which has been applied to several different conditions. Gowers mentions a case in which there were grayish-red spots at the junction of the white and gray matters, and in which the neuroglia was increased. There is also a condition in which, on the surface of the convolutions, there are small nodular projections, varying from a half to five or more millimetres in diameter. Single nodules of this sort are not uncommon; sometimes they are abundant. So far as is known no symptoms are produced by them.

(2) **Diffuse sclerosis,** which may involve an entire hemisphere, or a single lobe, in which case the term *sclérose lobaire* has been applied to it by the French. It is not an important condition in general medical practice, but occurs most frequently in idiots and imbeciles. In extensive cortical sclerosis of one hemisphere the ventricle is usually dilated.* The symptoms of this condition depend upon the region affected. There may be a considerable extent of sclerosis without symptoms or without much mental impairment. In a majority of cases there is hemiplegia or diplegia with imbecility or idiocy.

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* In my monograph on Cerebral Palsies of Children I have given a description of the distribution of the sclerosis in ten specimens in the museum at the Elwyn Institution.

(3) **Tuberous Sclerosis.**—In this remarkable form, which is also known as hypertrophic sclerosis, there are on the convolutions areas, projecting beyond the surfaces, of an opaque white color and exceedingly firm. The sclerosis may not disturb the symmetry of the convolution, but simply cause a great enlargement, increase in the density, and a change in the color.

These three forms are not of much practical interest except in asylum and institution work. The last variety forms a well-characterized disease of considerable importance, namely:

(4) Insular Sclerosis (*Sclérose en plaques*).

Definition.—A chronic affection of the brain and cord, characterized by localized areas in which the nerve elements are more or less replaced by connective tissue. This may occur in the brain or cord alone, more commonly in both.

Etiology.—This is obscure. Kahler, Marie, and others assign great importance to the infectious diseases, particularly scarlet fever. It is found most commonly in young persons, and cases are not uncommon in children, in whom Pritchard states that more than 50 cases have been reported. Sachs has recently reviewed the whole subject (Jour. of Nerv. and Mental Diseases, 1898).

Morbid Anatomy.—The sclerotic areas are widely distributed through the brain and cord, and cases limited to either part alone are almost unknown. The grayish-red areas are scattered indifferently through the white and gray matter (E. W. Taylor). The patches are most abundant in the neighborhood of the ventricles, and in the pons, cerebellum, basal ganglia, and the medulla. The cord may be only slightly involved or there may be irregular areas in different regions. The cervical region is most often the seat of nodules. The nerve-roots and the branches of the cauda equina are often attacked. Histologically in the sclerosed patches there is very marked proliferation of the neuroglia, the fibres of which are denser and firmer. The gradual growth destroys the medulla of the nerves, but the axis cylinders persist in a remarkable way. There is as a consequence relatively little secondary degeneration of nerve tracts.

Symptoms.—The onset is slow and the disease is chronic. Feebleness of the legs with irregular pains and stiffness are among the early symptoms. Indeed, the clinical picture may be that of spastic paraplegia with great increase in the reflexes. The following are the most important features:

(*a*) *Volitional Tremor or So-called Intention Tremor.*—There is no paralysis of the arms, but on attempting to pick up an object there is trembling or rapid oscillation. A patient may be unable to lift even a glass of water to the mouth. The tremor may be marked in the legs and in the head, which shakes as he walks. When the patient is recumbent the muscles may be perfectly quiet. On attempting to raise the head from the pillow, trembling at once comes on. (*b*) *Scanning Speech.*—The words are pronounced slowly and separately, or the individual syllables may be accentuated. This staccato or syllabic utterance is a common feature. (*c*) *Nys-*

tagmus, a rapid oscillatory movement of both eyes, constitutes an important symptom.

Sensation is unaffected in a majority of the cases. Optic atrophy sometimes occurs, but not so frequently as in tabes. The sphincters, as a rule, are unaffected until the last stages. Mental debility is not uncommon. Remarkable remissions occur in the course of the disease, in which for a time all the symptoms may improve. Vertigo is common, and there may be sudden attacks of coma, such as occur in general paresis.

The symptoms, on the whole, are extraordinarily variable, corresponding to the very irregular distribution of the nodules.

The *diagnosis* in well-marked cases is easy. Volitional tremor, scanning speech, and nystagmus form a characteristic symptom-group. With this there is usually more or less spastic weakness of the legs. Paralysis agitans, certain cases of general paresis, and occasionally hysteria may simulate the disease very closely. If the case is not seen until near the end the diagnosis may be impossible. Buzzard holds that of all organic diseases of the nervous system disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria. The points to be relied upon in the differentiation are, in order of importance, the nystagmus, the bladder disturbances, and the volitional tremor. The tremor in hysteria is not volitional.

Much more puzzling, however, are the instances of *pseudo-sclérose en plaques*, which have been described by Westphal. French writers regard them as instances of hysterical tremor. In children the condition may with difficulty be separated from Friedreich's ataxia.

The *prognosis* is unfavorable. Ultimately, the patient, if not carried off by some intercurrent affection, becomes bedridden.

Treatment.—No known treatment has any influence on the progress of sclerosis of the brain. Neither the iodides nor mercury have the slightest effect, but a prolonged course of nitrate of silver may be tried, and arsenic is recommended.

III. CHRONIC DIFFUSE MENINGO-ENCEPHALITIS

(*Dementia Paralytica; General Paresis*).

Definition.—A chronic, progressive meningo-encephalitis associated with psychical and motor disturbances, finally leading to dementia and paralysis.

Etiology.—Males are affected much more frequently than females. It occurs chiefly between the ages of thirty and fifty-five. Heredity is a factor in only a few instances. An overwhelming majority of the cases are in married people. Statistics show that it is more common in the lower classes of society, but in this country in general medical practice the disease is certainly more common in the well-to-do classes. An important predisposing cause is "a life absorbed in ambitious projects with all its strongest mental efforts, its long-sustained anxieties, deferred hopes, and straining expectation" (Mickle). The habits of life so frequently seen in

active business men in our large cities, and well expressed by the phrase "burning the candle at both ends," strongly predispose to the disease. The important individual factor is syphilis, which is an antecedent in from 70 to 90 per cent of all cases. To this disease dementia paralytica and tabes dorsalis are so closely related that Fournier describes them under the heading *Les Affections Parasyphilitiques*. His recent work, with this title, is full of interesting details gleaned from an enormous experience. He suggests that these two disorders may be not merely diverse expressions of one and the same morbid entity, but that they possibly may be one and the same disease.

Morbid Anatomy.—The essential histological changes in the cerebral cortex are thus summarized by Bevan Lewis: (1) A stage of inflammatory change in the tunica adventitia of the arteries with excessive nuclear proliferation, profound changes in the vascular channels, and trophic changes induced in the tissues around.

(2) A stage of extraordinary development of the lymph-connective system of the brain, with a parallel degeneration and disappearance of nerve elements and the axis cylinders of which they are denuded.

(3) A stage of general fibrillation with shrinking and extreme atrophy of the parts involved.

The macroscopical changes are: Increase in the cerebro-spinal fluid, œdema of the pia, and thickening and opacity of the meninges, which are adherent in places and tear the cortex on removal. The dura is sometimes thickened, and pachymeningitis hæmorrhagica interna may be present.

The convolutions are atrophied, usually in a marked degree, and in consequence the brain looks small. This is particularly noticeable in the frontal and parietal regions. Flechsig suggests, from his own experience and that of Tuczek, that the different types met with are dependent upon the localization of the malady in given cases, predominantly in the anterior or in the posterior "association centre." On section the brain cuts with firmness. In extreme cases the gray matter may be obscurely outlined. The grade of sclerosis varies much in different cases. The white matter may be firmer in consistence, but it does not show such important changes. The ventricles are dilated and the ependyma is extremely granular. In addition, there are frequently areas of softening or hæmorrhage associated with chronic arterio-sclerosis.

The degenerative changes are not limited to the cortex, but also invade subcortical regions and the spinal cord. In the spinal cord changes are almost constantly found, usually sclerosis of the dorsal fasciculi, either alone or, more commonly, with involvement of the lateral.

Symptoms.—(*a*) *Prodromal Stage.*—This is of variable duration, and is characterized by a general mental state which finds expression in symptoms trivial in themselves but important in connection with others. Irritability, inattention to business amounting sometimes to indifference or apathy, and sometimes a *change in character* marked by acts, which may astonish the friends and relatives, may be the first indications. There may be unaccountable fatigue after moderate physical or mental exertion. Instead of apathy or indifference there may be an extraordinary degree of

physical and mental restlessness. The patient is continually planning and scheming, or may launch into extravagances and speculation of the wildest character. A common feature at this period is the display of an unbounded egoism. He boasts of his personal attainments, his property, his position in life, or of his wife and children. Following these features are important indications of moral perversion, manifested in offences against decency or the law, many of which acts have about them a suspicious effrontery. Forgetfulness is common, and may be shown in inattention to business details and in the minor courtesies of life. At this period there may be no motor phenomena. The onset of the disease is usually insidious, although cases are reported in which epileptiform or apoplectiform seizures were the first symptoms. Among the early motor features are tremor of the tongue and lips in speaking, slowness of speech and hesitancy, inequality of the pupils, and the Argyll Robertson pupil.

(*b*) *Second Stage.*—This is characterized in brief by mental exaltation or excitement and a progress in the motor symptoms. "The intensity of the excitement is often extreme, acute maniacal states are frequent; incessant restlessness, obstinate sleeplessness, noisy, boisterous excitement, and blind, uncalculating violence especially characterize such states" (Lewis). It is at this stage that the delusion of grandeur becomes marked and the patient believes himself to be possessed of countless millions or to have reached the most exalted sphere possible in profession or occupation. This expansive delirium, as it is called, is, however, not characteristic, as was formerly supposed, of paralytic dementia. Besides, it does not always occur, but in its stead there may be marked melancholia or hypochondriasis, or, in other instances, alternate attacks of delirium and depression.

The facies has a peculiar stolidity, and in speaking there is marked tremulousness of the lips and facial muscles. The tongue is also tremulous, and may be protruded with difficulty. The speech is slow, interrupted, and blurred. Writing becomes difficult on account of unsteadiness of the hand. Letters, syllables, and words may be omitted. The subject matter of the patient's letters gives valuable indications of the mental condition. In many instances the pupils are unequal, irregular, sluggish, sometimes large. Important symptoms in this stage are apoplectiform seizures and paralysis. There may be slight syncopal attacks in which the patient turns pale and may fall. Some of these are *petit mal.* In the true apoplectiform seizure the patient falls suddenly, becomes unconscious, the limbs are relaxed, the face is flushed, the breathing stertorous, the temperature increased, and death may occur. The epileptic seizures are more common than the apoplectiform and may occur in the disease. A definite aura is not uncommon. The attack usually begins on one side and may not spread. There may be twitchings either in the facial or brachial muscles. Typical Jacksonian epilepsy may occur. In a case which died recently under my care, these seizures were among the early symptoms and the disease was regarded as cerebral syphilis. Paralysis, either monoplegic or hemiplegic, may follow these epileptic seizures, or may come on with great suddenness and be transient. In this stage the gait becomes impaired, the patient trips readily, has difficulty in going up or down stairs, and the walk

may be spastic or occasionally tabetic. This paresis may be progressive. The knee-jerk is usually increased. Bladder or rectal symptoms gradually develop. The patient becomes helpless, bedridden, and completely demented, and unless care is taken may suffer from bedsores. Death occurs from exhaustion or from some intercurrent affection. The absence of pain reaction on pressure upon the ulnar nerve behind the elbow (Biernacki's symptom) is apparently not of any special value. The spinal-cord features of dementia paralytica may come on with or precede the mental troubles; in 80 per cent of the cases they follow them. There are cases in which one is in doubt for a time whether the symptoms indicate tabes or dementia paralytica, and it is well to bear in mind that every feature of pre-ataxic tabes may exist in the early stage of general paresis.

Diagnosis.—The recognition of the disease in the earliest stage is extremely difficult, as it is often impossible to decide that the slight alteration in conduct is anything more than one of the moods or phases to which most men are at times subject. The following description by Folsom is an admirable presentation of the diagnostic characters of the early stage of the disease: "It should arouse suspicion if, for instance, a strong, healthy man, in or near the prime of life, distinctly not of the 'nervous,' neurotic, or neurasthenic type, shows some loss of interest in his affairs or impaired faculty of attending to them; if he becomes varyingly absent-minded, heedless, indifferent, negligent, apathetic, inconsiderate, and, although able to follow his routine duties, his ability to take up new work is, no matter how little, diminished; if he can less well command mental attention and concentration, conception, perception, reflection, judgment; if there is an unwonted lack of initiative, and if exertion causes unwonted mental and physical fatigue; if the emotions are intensified and easily change, or are excited readily from trifling causes; if the sexual instinct is not reasonably controlled; if the finer feelings are even slightly blunted; if the person in question regards with a placid apathy his own acts of indifference and irritability and their consequences, and especially if at times he sees himself in his true light and suddenly fails again to do so; if any symptoms of cerebral vaso-motor disturbances are noticed, however vague or variable."

There are cases of cerebral syphilis which closely simulate dementia paralytica. The mode of onset is important, particularly since paralytic symptoms are usually early in syphilis. The affection of the speech and tongue is not present. Epileptic seizures are more common and more liable to be cortical or Jacksonian in character. The expansive delirium is rare. While symptoms of general paresis are not common in connection with the development of gummata or definite gummatous meningitis, there are, on the other hand, instances of paresis which follow syphilitic infection so closely that an etiological connection between the two must be acknowledged. Post mortem in such cases there may be nothing more than a general arterio-sclerosis and diffuse meningo-encephalitis, which may present nothing distinctive, but the lesions, nevertheless, may be caused by the syphilitic virus. There are certain forms of lead encephalopathy which resemble general paresis, and, considering the association of plumbism with arterio-sclerosis, it is not unlikely that the anatomical substratum of the

disease may result from this poison. Tumor may sometimes simulate progressive paresis, but in the former the signs of general increase of the intracranial pressure (pain in the head, choked disks, slowing of the pulse-rate, projectile vomiting) are usually present.

Prognosis.—The disease rarely ends in recovery. As a rule the progress is slowly downward and the case terminates in a few years, although it is occasionally prolonged ten or fifteen years.

Treatment.—The only hope of permanent relief is in the cases following syphilis, which should be placed upon large doses of iodide of potassium. Careful nursing and the orderly life of an asylum are the only measures necessary in a great majority of the cases. For sleeplessness and the epileptic seizures bromides may be used. Prolonged remissions, which are not uncommon, are often erroneously attributed to the action of remedies. Active treatment in the early stage by wet-packs, cold to the head, and systematic massage have been followed by temporary improvement.

IV. DIFFUSE AND FOCAL DISEASES OF THE SPINAL CORD.

I. TOPICAL DIAGNOSIS.

We have seen that a lesion involving a definite part of the gray matter of the lower motor segment is accompanied by loss of the power to perform certain definite movements. A disease, such as anterior polio-myelitis, which is confined to the gray matter, gives as its only symptom a characteristic lower-segment paralysis. The muscles paralyzed reveal the seat of the lesion. In many instances a transverse section of the spinal cord is involved to a greater or less extent; if complete, there is lower-segment paralysis at the level of the lesion. If the muscles so paralyzed are the same on the two sides of the body, the lesion is strictly transverse, for, obviously, if the cord is involved higher on one side than on the other the paralyzed muscles will vary accordingly. Besides the paralysis due to involvement of the lower segment, the muscles whose centres are below the lesion may also be paralyzed by the involvement of the upper segment in the pyramidal tract, and present all the characteristics of such a paralysis. The degree of the paralysis depends upon the intensity of the lesion of the pyramidal tract, and varies from a slight weakness in the flexion of the ankle to an absolute paralysis of all the muscles below the lesion. The sphincter muscles of the bladder and rectum are also often paralyzed.

Sensory symptoms are usually less prominent, but when the spinal cord is much diseased there is a dulling of sensation all over the body below the lesion. The upper border of disturbed sensation often indicates the level of the disease, especially when this is in the thoracic region, where the corresponding motor paralysis is not easy to demonstrate. It is to be noted that the anæsthesia does not reach quite to the level of the lesion; thus if the fifth thoracic segment be involved, the anæsthesia will include the

area supplied by the sixth segment, but not that supplied by the fifth. This is due to the overlapping of the areas. There is often a narrow zone of hyperæsthesia above the anæsthetic region.

When the transverse lesion is complete and the lower part of the cord is cut off from all influence from above, there is complete sensory and motor paralysis, and the deep reflexes instead of being exaggerated are lost.

The different reflexes are dependent upon different levels of the cord (see Starr's table, p. 905), and their absence or presence may be important localizing symptoms.

Unilateral Lesions.—The motor symptoms which follow lesions confined to one half of the cross-section of the spinal cord follow the same rules as those given for transverse lesions, except that they are confined to one side of the body—that is, they are on the same side as the lesion.

The sensory symptoms are peculiar. On the side corresponding to the disease—the paralyzed side—there is anæsthesia corresponding to the segment of the cord involved; above this there is a narrow zone of hyperæsthesia, but below this there is no diminution in the senses of touch, pain, or temperature; indeed, there is often hyperæsthesia. The muscular sense, however, is impaired. On the side opposite to the lesion there may be complete loss of the sense of touch, pain, and temperature, or it may only involve one or two of these, pain and temperature usually being associated.

The following table, slightly modified from Gowers, illustrates the distribution of these symptoms in a complete hemi-lesion of the cord:

Cord.

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia. Lower segment paralysis with atrophy.	Lesion.	
Upper segment paralysis. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened and then increased. Temperature raised.		Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

It is only in exceptional cases that all these features are met with, for they vary with its extent and intensity.

This combination of symptoms was first recognized by Brown-Séquard, after whom it has been named. It may follow tumors, stab-wounds, fracture and caries of the spine, and it is not infrequently associated with syringomyelia and hæmorrhages into the cord.

The explanation of the disturbance in sensation is not satisfactory, and cannot be until our knowledge of the paths of sensory conduction is more accurate. These cases have convinced most clinicians that in man the paths for touch, pain, and temperature cross in the middle line soon after entering the spinal cord, and proceed toward the brain in the opposite side, while that for muscular sense remains in the dorsal columns of the

same side. We have seen that anatomy lends some support to this view, and this is the explanation that is usually given. The experiments on animals have thrown some doubt on this view, especially those of Mott on monkeys, which seem to indicate that the sensory paths for the most part remain on the same side of the cord.

II. AFFECTIONS OF THE BLOOD-VESSELS.

1. Congestion.

Apart from actual myelitis, we rarely see post mortem evidences of congestion of the spinal cord, and when we do, it is usually limited either to the gray matter or to a definite portion of the organ. There is necessarily, from the posture of the body post mortem, a greater degree of vascularity in the dorsal portion of the cord. The white matter is rarely found congested, even when inflamed; in fact, it is remarkable how uniformly pale this portion of the cord is. The gray matter often has a reddish-pink tint, but rarely a deep reddish hue, except when myelitis is present. If we know little anatomically of conditions of congestion of the cord, we know less clinically, for there are no features in any way characteristic of it.

2. Anæmia.

So, too, with this state. There may be extreme grades of anæmia of the cord without symptoms. In chlorosis and pernicious anæmia there are rarely symptoms pointing to the cord, and there is no reason to suppose that such sensations as heaviness in the limbs and tingling are especially associated with anæmia.

There are, however, some very interesting facts with reference to the profound anæmia of the cord which follows ligature of the aorta. In experiments made in Welch's laboratory by Herter, it was found that within a few moments after the application of the ligature to the aorta paraplegia came on. Paralysis of the sphincters developed, but less rapidly. This condition is of interest in connection with the occasional rapid development of a paraplegia after profuse hæmorrhage, usually from the stomach or uterus. It may come on at once or at the end of a week or ten days, and is probably due to an anatomical change in the nerve elements similar to that produced in Herter's experiments. The degeneration of the dorsal columns of the cord in pernicious anæmia has already been described.

3. Embolism and Thrombosis.

Blocking of the spinal arteries by emboli rarely occurs. It may be produced experimentally, and Money found that it was associated with choreiform movements. Thrombosis of the smaller vessels in connection with endarteritis plays an important part in many of the acute and chronic changes in the cord.

Extravasation — i/

4. Endarteritis.

It is remarkable how frequently in persons over fifty the arteries of the spinal cord are found sclerotic. The following forms may be met with: (1) A nodular peri-arteritis or endarteritis associated with syphilis and sometimes with gummata of the meninges; (2) an arteritis obliterans, with great thickening of the intima and narrowing of the lumen of the vessels, involving chiefly the medium and larger-sized arteries. Miliary aneurisms or aneurisms of the larger vessels are rarely found in the spinal cord. In the classical work of Leyden but a single instance of the latter is mentioned.

5. Hæmorrhage into the Spinal Membranes; Hæmatorrhachis.

In meningeal apoplexy, as it is called, the blood may be between the dura mater and the spinal canal—extra-meningeal hæmorrhage—or within the dura mater—intra-meningeal hæmorrhage.

(*a*) *Extra-meningeal hæmorrhage* occurs usually as a result of trauma. The exudation may be extensive without compression of the cord. The blood comes from the large plexuses of veins which may surround the dura. The rupture of an aneurism into the spinal canal may produce extensive and rapidly fatal hæmorrhage.

(*b*) *Intra-meningeal hæmorrhage* is rather more common, but is rarely extensive from causes acting directly on the spinal meninges themselves. Scattered hæmorrhages are not infrequent in the acute infectious fevers, and I have twice, in malignant small-pox, seen much effusion. Bleeding occurs also in death from convulsive disorders, such as epilepsy, tetanus, and strychnia poisoning. The most extensive hæmorrhages occur in cases in which the blood comes from rupture of an aneurism at the base of the brain, either of the basilar or vertebral artery. In several cases of this kind I have found a large amount of blood in the spinal meninges. In ventricular apoplexy the blood may pass from the fourth ventricle into the spinal meninges. There is a specimen in the medical museum of McGill College of the most extensive intraventricular hæmorrhage, in which the blood passed into the fourth ventricle, and descended beneath the spinal arachnoid for a considerable distance. On the other hand, hæmorrhage into the spinal meninges may possibly ascend into the brain.

The *symptoms* in moderate grades may be slight and indefinite. In the non-traumatic cases the hæmorrhage may either come on suddenly or after a day or two of uneasy sensations along the spine. As a rule, the onset is abrupt, with sharp pain in the back and symptoms of irritation in the course of the nerves. There may be muscular spasms, or paralysis may come on suddenly, either in the legs alone or both in the legs and arms. In some instances the paralysis develops more slowly and is not complete. There is no loss of consciousness, and there are no signs of cerebral disturbance. The clinical picture naturally varies with the site of the hæmorrhage. If in the lumbar region, the legs alone are involved, the reflexes may be abolished, and the action of the bladder and rectum is impaired. If in the thoracic region, there is more or less complete paraplegia, the reflexes are

usually retained, and there are signs of disturbance in the thoracic nerves, such as girdle sensations, pains, and sometimes eruption of herpes. In the cervical region the arms as well as the legs may be involved; there may be difficulty in breathing, stiffness of the muscles of the neck, and occasionally pupillary symptoms.

The prognosis depends much upon the cause of the hæmorrhage. Recovery may take place in the traumatic cases, and in those associated with the infectious diseases.

6. Hæmorrhage into the Spinal Cord (*Hæmatomyelia*).

It is more common in males than in females, and at the middle period of life. The cases have followed either cold and exposure or overexertion, and, most frequently of all, traumatism. It is most frequent in the lower cervical region, the most common site for dislocation and fracture of the spine. It occurs also in tetanus and convulsions. Hæmorrhage into the cord may follow injuries of the spinal column, gun-shot wounds, etc., even when the cord itself has not been touched (H. Cushing). Hæmorrhage may be associated with tumors, with syringo-myelia, or with myelitis; it is often difficult to determine whether the case is one of primary hæmorrhage with myelitis, or myelitis with a secondary hæmorrhage.

The *anatomical condition* is very varied. The cord may be enlarged at the site of the hæmorrhage, and occasionally the white substance may be lacerated and blood may escape beneath the meninges. The extravasation is chiefly in the gray matter, and may be limited or focal, or very diffuse, extending a considerable distance in the cord. In a case which occurred at the Montreal General Hospital under Wilkins the hæmorrhage occupied a position opposite the region of the fifth and sixth cervical nerves and on transverse section the cord was occupied by a dark-red clot measuring 12 by 5 mm., around which the white substance formed a thin, ragged wall. The clot could be traced upward as far as the second cervical, and downward as far as the fourth thoracic segment.

The sudden onset of the *symptoms* is the most characteristic feature in hæmatomyelia. The loss of power necessarily varies with the locality affected. If in the cervical region, both arms and legs may be involved; but if in the thoracic or lumbar, there is only paraplegia. There is usually loss of sensation, and at first loss of reflexes. Myelitis frequently develops and becomes extensive, with fever and trophic changes. The condition may rapidly prove fatal; in other instances there is gradual recovery, often with partial paralysis.

The diagnosis may be made in some instances, particularly those in which the onset is sudden after injury, but there is great difficulty in differentiating hæmorrhagic myelitis from certain cases of hæmorrhage into the spinal meninges.

Pain "the most obscure character about the knee
Elbow" or other joints, without swelling as a rule. Pain &
Swelling in the muscles, specially pain & wasting are
are the most common symptoms. Headache, giddiness
& paralysis are less frequent. Paraplegia occurred
in 15 per cent of 1/2 or Smith's cases & in 6 per cent of the
Blackwall Tunnel cases.

Deaths were, 12 of 76 cases at the St
Lewis Bridge, 3 in 110 at Bootle
Bridge. In the recent important work
of the Forth Bridge & the Blackwall Tunnel
there were no fatalities from these causes.

The most successful treatment is by recompression.
A medical man will should be provided at the works, well
headed & pillow with bromides &c to stop the pain & relieve
the symptoms. Morphia may be required.

Warrington. Lancet. 12.2.00

7. Caisson Disease (*Diver's Paralysis; Compressed Air Disease*).

This remarkable affection, found in divers and in workers in caissons, is characterized by a paraplegia, more rarely a general palsy, which supervenes on returning from the compressed atmosphere to the surface.

The disease has been carefully studied by the French writers, by Leyden and Schultze in Germany, and in this country particularly by A. H. Smith. It has been made the subject of a special monograph by Snell. The pressure must be more than that of three atmospheres. The symptoms are especially apt to come on if the change from the high to the ordinary atmospheric pressure is quickly made. They may supervene immediately on leaving the caisson, or they may be delayed for several hours. In the mildest form there are simply pains about the knees and in the legs, often of great severity, and occurring in paroxysms. Abdominal pain and vomiting are not uncommon. The legs may be tender to the touch, and the patient may walk with a stiff gait. Dizziness and headache may accompany these neuralgic symptoms, or may occur alone. More commonly in the severe form there is paralysis both of motion and sensation, usually a paraplegia, but it may be general, involving the trunk and arms. Monoplegia and hemiplegia are rare. In the most extreme instances the attacks resemble apoplexy; the patient rapidly becomes comatose and death occurs in a few hours. In the case of paraplegia the outlook is usually good, and the paralysis may pass off in a day, or may continue for several weeks or even for months.

The explanation of this condition is by no means satisfactory. Several careful autopsies have been made. In Leyden's case death occurred on the fifteenth day, and in the thoracic portion of the cord there were numerous foci of hæmorrhages and signs of an acute myelitis. In Schultze's case death occurred in two and a half months, and a disseminated myelitis was found in the thoracic region. In both cases there were fissures, and appearances as if tissue had been lacerated. In a case examined on the third day (Ziegler's Beiträge, 1892) this condition of fissuring and laceration was found. It has been suggested that the symptoms are due to the liberation in the spinal cord of bubbles of nitrogen which have been absorbed by the blood under the high pressure, and the condition found at the autopsies just referred to is held to favor this view.

A large majority of the cases recover. The severe neuralgic pains often require morphia. Inhalations of oxygen and the use of compressed air have been advised. When paraplegia develops the treatment is similar to that of other forms. In all caisson work care should be exercised that the time in passing through the lock from the high to the ordinary pressure be sufficiently prolonged. Snell lays less stress on this than on the proper ventilation of the caisson.

III. COMPRESSION OF THE SPINAL CORD

(*Compression Myelitis*).

Definition.—Interruption of the functions of the cord by slow compression.

Etiology.—Caries of the spine, new growths, aneurism, and parasites are the important causes of slow compression. Caries, or Pott's disease, as it is usually called, after the surgeon who first described it, is in the great majority of instances a tuberculous affection. In a few cases it is due to syphilis and occasionally to extension of disease from the pharynx. It is most common in early life, but may occur after middle age. It follows trauma in a few cases. Compression occasionally results from aneurism of the thoracic aorta or the abdominal aorta, in the neighborhood of the cœliac axis.

Malignant growths frequently cause a compression paraplegia. A retroperitoneal sarcoma or the lymphadenomatous growths of Hodgkin's disease may invade the vertebræ. More commonly, however, the involvement is secondary to scirrhus of the breast.

Of parasites, the echinococcus and the cysticercus occasionally occur in the spinal canal. For a masterly consideration of the whole question, particularly from a surgical standpoint, Kocher's monograph is all-important (Mitt. a. d. Grenzgebiet. der Chir. u. d. Med., 1896, Bd. i).

Symptoms.—These may be considered as they affect the bones, the nerves, and the cord.

(1) **Vertebral.**—In malignant diseases and in aneurism, erosion of the bodies may take place without producing any deformity of the spine. Fatal hæmorrhage may follow erosion of the vertebral artery. In caries, on the other hand, it is the rule to find more or less deformity, amounting often to angular curvature. The compression is largely due to the thickening of the dura and the presence of caseous and inflammatory products between this membrane and the bone. The compression is rarely produced directly by the bone. Pain is a constant and, in the case of aneurism and tumor, an agonizing feature. In caries, the spinal processes of the affected vertebræ are tender on pressure, and pain follows jarring movements or twisting of the spine. There may be extensive tuberculous disease without much deformity, particularly in the cervical region.

(2) **Nerve-root Symptoms.**—These result from compression of the nerve roots as they pass out between the vertebræ. A cervico-brachial neuralgia may be an early symptom. It is remarkable how frequently, even in extensive caries, they escape and the patient does not complain of radiating pains in the distribution of the nerves from the affected segment. Pains are more common in cancer of the spine secondary to that of the breast, and in such cases may be agonizing. There may be acutely painful areas—the *anæsthesia dolorosa*, in regions of the skin which are anæsthetic to tactile and painful impressions. Trophic disturbances may occur, particularly herpes. In the cervical or lumbar regions pressure on the ventral roots may give rise to wasting of the muscles supplied by the affected nerves.

(3) **Cord Symptoms.**—(*a*) *Cervical Region.*—Not infrequently the caries is high up between the axis and the atlas or between the latter and the occipital bone. In such instances a retropharyngeal abscess may be present, giving rise to difficulty in swallowing. There may be spasm of the cervical muscles, the head may be fixed, and movements may either be impossible or cause great pain. In a case of this kind in the Montreal General Hospital movement was liable to be followed by transient, instantaneous paralysis of all four extremities, owing to compression of the cord. In one of these attacks the patient died.

In the lower cervical region there may be signs of interference with the cilio-spinal centre and dilatation of the pupils. Occasionally there is flushing of the face and ear of one side or unilateral sweating. Deformity is not so common, but healing may take place with the production of a callus of enormous breadth, with complete rigidity of the neck.

(*b*) *Thoracic Region.*—The deformity is here more marked and pressure symptoms are more common. The time of onset of the paralysis varies very much. It may be an early symptom, even before the curvature is manifest. More commonly it is late, occurring many months after the curvature has developed. The paraplegia is slow in its development; the patient at first feels weak in the legs or has disturbance of sensation, numbness, tingling, pins and needles. The girdle sensation may be marked, or severe pains in the course of the intercostal nerves. Motion is, as a rule, more quickly lost than sensation. Bastian's symptom—abolition of the reflexes—is rarely met with in compression from caries. Finally, there is complete interruption with the production of paraplegia, usually of the spastic type, with exaggeration of the reflexes. This may persist for months, or even for more than a year, and recovery still be possible.

(*c*) *Lumbar Region.*—In the lower dorsal and lumbar regions the symptoms are practically the same, but the sphincter centres are involved and the reflexes are not exaggerated.

Diagnosis.—Caries is by far the most frequent cause of slow compression of the cord, and when there are external signs the recognition is easy. There are cases in which the exudation in the spinal canal between the dura and the bone leads to compression before there are any signs of caries, and if the root symptoms are absent it may be extremely difficult to arrive at a diagnosis. Janeway has called attention to persistent lumbago as a symptom of importance in masked Pott's disease, particularly after injury. Brown-Séquard's paralysis is more common in tumor and in injuries than in caries. Pressure on the nerve roots, too, is less frequent in caries than in malignant disease. The cervical form of pachymeningitis also produces a pressure paralysis, the symptoms of which have already been detailed. Pressure from secondary carcinoma is naturally suggested when spinal symptoms follow within a few years after an operation for cancer of the breast. In paraplegia following tumor of the vertebra secondary to cancer of the breast, and in the erosion of the spine by retroperitoneal growths, the suffering is most intense. The condition has been well termed *paraplegia dolorosa.* I have seen 2 cases in which the breast tumor had not been recognized.

Treatment.—In compression by aneurism or tumor the condition is hopeless. In the former the pains are often not very severe, but in the latter morphia is always necessary. On the other hand, compression by caries is often successfully relieved even after the paralysis has persisted for a long period. When caries is recognized early, rest and support to the spine by the various methods now used by surgeons may do much to prevent the onset of paraplegia. When paralysis has developed, rest with extension gives the best hope of recovery. It is to be remembered that restoration may occur after compression of the cord has lasted for many months, or even more than a year. Cases have been cured by rest alone; the extradural and inflammatory products are absorbed and the caries heals. The most brilliant results in these cases have been obtained by suspension, a method introduced by J. K. Mitchell in 1826, and pursued with remarkable success by his son, Weir Mitchell. During my association with the Infirmary for Nervous Diseases I had numerous opportunities of witnessing the really remarkable effects of persistent suspension, even in apparently desperate and protracted cases. Mitchell's conclusions are that suspension should be employed early in Pott's disease; that used with care it enables us slowly to lessen the curve; that in these cases there must be, in some form, a replacement of the crumpled tissues; that unless there is great loss of power the use of the spine-car or chair of J. K. Mitchell enables suspension, especially in children, to be combined with some exercise; that no case of Pott's disease should be considered desperate without its trial; that suspension has succeeded after failures of other accepted methods; that the pull probably acts more or less directly on the cord itself, and that the gain is not explicable merely by obvious effects on the angular bone curve; that the methods of extension to be used in carious cases may be very varied, provided only we get active extension; that the plan and the length of time of extension must be made to conform to the needs, endurance, and sensation of the individual case. It may be months before there are any signs of improvement. In protracted cases, after suspension has been tried for months, laminectomy may be considered, and has in some instances been successful.

The general treatment of caries is that of tuberculosis—fresh air, good food, cod-liver oil, and arsenic. Counter-irritation in these instances is of doubtful value.

Lesions of the Cauda Equina and Conus Medullaris.

The spinal cord extends only to the second lumbar vertebra. Injury, tumors, and caries at or below this level involve not the cord itself, but the bundle of nerves known as the cauda equina and the terminal portion of the cord, the conus medullaris. Much attention has been given to lesions of this part. The whole subject is admirably discussed in Thorburn's work. Fractures and dislocations are common in the lumbo-sacral region, tumors not infrequently involve the filaments of the cauda equina, and some of the nerves may be entangled in the cicatrix of a spina bifida.

In a fracture or dislocation of the first lumbar vertebra the conus me-

dullaris may be compressed with the last sacral nerves given off from it. In a case reported by Kirchhoff there was laceration of the conus with complete paralysis of the bladder and rectum, a case which is held to favor the view that the ano-vesical centre in man is situated in this region of the cord. There are several instances on record in which injury of the cauda equina has produced paralysis of the bladder and rectum alone, sometimes with a slight patch of anæsthesia in the neighborhood of the coccyx or the perinæum. More commonly branches of the sacral or lumbar nerve roots are involved, producing an irregularly distributed motor and sensory paralysis in the legs. When the lumbar nerve roots from the second to the fifth are compressed, there is paralysis of the muscles of the legs, with the exception of the flexors of the ankles, the peronæi, the long flexors of the toes, and the intrinsic muscles of the feet, and loss of sensation in the front, inner and outer part of the thighs, the inner side of the legs, and the inner side of the foot. The sacral roots may alone be involved. Thus in a case which I have reported the patient fell from a bridge and had paralysis of the legs and of the bladder and rectum. When seen sixteen years after the injury, there was slight weakness, with wasting of the left leg; there was complete loss of the function in the ano-vesical and genital centres, and anæsthesia in a strip at the back part of the thigh (in the distribution of the small sciatic), and of the perinæum, scrotum, and penis. The urethra was also insensitive.

Starr's table and Head's figures, given in the general introduction, will be found useful in determining the nerve fibres and segments involved in these cases of injury of the cauda equina.

IV. TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

New growths may develop in the cord or in its membranes, or may extend into them from the spine. The first two alone will be considered. Occasionally lipoma and parasites occur in the extradural space. Within the dura fibromata, sarcomata, and syphilitic and tuberculous growths are most common. In the cord itself, and attached to the pia mater, the tuberculous, syphilitic, and gliomatous growths are most frequent. Of 50 cases of tumor of the spinal cord and its envelopes, analyzed by Mills and Lloyd, only 3 were parasitic. Of these, 26 were some form of neoplasm, of which sarcomata were most common, 5 were gummatous, and 4 tuberculous. Herter has recently reported 3 cases of solitary tubercle in the cord, and has analyzed others from the literature. Of 24 cases in which the age was given, 15 occurred between the ages of fifteen and thirty-five, and 5 before the fifth year. The tumor is most common in the dorsal and lumbar regions, and is usually met with in connection with tuberculous lesions elsewhere.

The anatomical effects of tumor are very varied. Slow compression is usually produced by growths external to the cord, and it is remarkable what a high grade of compression the cord will bear without serious inter-

ference with its functions. In cases of prolonged interruption ascending and descending degenerations occur. Tumors developing within the cord may lead to syringo-myelia. And, lastly, tumors not infrequently excite intense myelitis.

Symptoms.—These will naturally vary a good deal with the segment involved and with the degree of pressure and the extent of implication of the nerve roots.

Within the cord the symptoms are those of a gradually progressing paraplegia, which may at first have the picture of a Brown-Séquard paralysis. Atrophy follows the involvement of the ventral cornua, and vasomotor disturbances may be marked. The reflexes are lost at the level of the lesion, but if this be in the thoracic cord, the reflexes are retained in the legs. The symptoms are apt to be complicated with those of acute or subacute myelitis, which may completely alter the clinical picture. Tumors of the spinal membranes are characterized by the early onset and persistence of the root symptoms, which consist of radiating pains, the girdle sensation, and hyperæsthesia, or anæsthesia in various portions of the trunk. There may even be severe pain in the anæsthetic areas. Irritation of the motor roots may cause spasm of the muscles supplied, or wasting with paralysis. The paraplegia supervenes some time after the occurrence of the root symptoms. In the thoracic region the level of the growth is usually accurately defined by the level of the pain and the condition of the reflexes.

The diagnosis of tumor within the cord is sometimes easy, the characteristic features being the constancy and severity of the root symptoms at the level of the growth and the progressive paralysis. Caries may cause identical symptoms, but the radiating pains are rarely so severe. Cervical meningitis simulates tumor very closely, and in reality produces identical effects, but the very slow progress and the bilateral character from the outset may be sufficient to distinguish it.

In chronic transverse myelitis the symptoms may, according to Gowers, simulate tumor very closely and present radiating pains, a sense of constriction, and progressive paralysis.

The nature of the tumor can rarely be indicated with precision. With a marked syphilitic history gumma may naturally be suspected, and with coexisting tuberculous disease a solitary tubercle.

Treatment.—If the possibility of syphilitic infection is present the iodide of potassium should be given in large and increasing doses. For the severe pains counter-irritation is sometimes beneficial, particularly the thermo-cautery; morphia is, however, often necessary.

In a few instances tumors of the cord or of the membranes are amenable to surgical treatment. The removal by Horsley of a growth from the spinal membranes was one of the most brilliant of recent operations.

Abscess of the cord is a rare lesion, of which only 3 or 4 cases have been described, all metastatic. It may occur without meningitis.

V. SYRINGOMYELIA.

Definition.—A gliomatous new formation about the central canal of the spinal cord, with cavity formation.

Etiology and Morbid Anatomy.—Syringomyelia must be distinguished from dilatation of the central canal—hydromyelus—slight grades of which are not very uncommon either as a congenital condition or as a result of the pressure of tumors. The cavity of syringomyelia has a variable extent in the cord, sometimes running the entire length, but in many cases involving only the cervical and thoracic regions or a more limited area. It is usually in the dorsal portion of the cord and may extend only into one dorsal cornu. The transverse section may be oval or circular or narrow and fissure-like. It varies at different levels. The condition is now regarded as a *gliosis*, a development of embryonal neurogliar tissue in which hæmorrhage or degeneration takes place with the formation of cavities.

Of 190 cases, 133 were in men, 57 in women (Schlesinger). A large majority of the cases begin before the thirtieth year. The disease has been met with in three members of the same family.

Symptoms.—The clinical features are extremely complex. In the classical form there are irregular pains, chiefly in the cervical region; muscular atrophy develops, which may be confined to the arms, or sometimes extends to the legs. The reflexes are increased and a spastic condition develops in the legs. Ultimately the clinical picture may be that of an amyotrophic lateral sclerosis. The tactile sensation is usually intact and the muscular sense is retained, but painful and thermic sensations are not recognized, or there may be in rare instances complete anæsthesia of the skin and of the mucous membranes (Dejerine). This combination of loss of painful and thermic sensations with paralysis of an amyotrophic type is regarded as pathognomonic of the disease. The special senses are usually intact and the sphincters uninvolved. Trophic troubles are not uncommon. Owing to the loss of the pain and heat sensations, the patients are apt to injure themselves. Scoliosis also may be present in these cases. The loss of painful and thermic impressions is due to the fact that these pass to the brain in the peri-ependymal gray matter, particularly that portion in the dorsal roots, which is almost constantly involved in syringomyelia. The tactile sensation is retained because the postero-lateral column is uninvolved.

Schlesinger, in his recent monograph (1895), recognizes the following types: (1) With the classical features above described, which may begin in the cervical or lumbar regions: (2) a motor type, with the picture of an amyotrophic or a spastic paralysis—the sensation may be undisturbed for years; (3) with predominant sensory features, simulating hysterical hemiplegia, or with general pain and temperature anæsthesia; (4) with pronounced trophic disturbances—to this type belong the cases described as Morvan's disease, an affection characterized by neuralgic pains, cutaneous anæsthesia, and painless, destructive whitlows; and (5) the tabetic type, either a combination of the symptoms of tabes in the lower, and of

syringomyelia in the upper extremities, or a pure tabetic symptom-complex, due to invasion by the gliosis of the dorsal columns (Oppenheim). Arthropathies occur in about 10 per cent of the cases.

In typical cases the *diagnosis* is easy. The combination of an amyotrophic paralysis, the picture of progressive muscular atrophy of the Aran-Duchenne type, with retention of tactile and loss of thermic and painful sensation, is probably pathognomonic of the disease. Of affections with which it may be confounded, anæsthetic leprosy is the most important, since the anæsthesia and the wasting may closely simulate it; but, as a rule, in leprosy trophic changes are more or less marked. There is often loss of phalanges and there is no characteristic dissociation of sensory impressions.

VI. ACUTE MYELITIS.

Etiology.—Acute myelitis results from many causes, and may affect the cord in a limited or extended portion—the gray matter chiefly, or the gray and white matter together. It is met with: (*a*) As an independent affection following exposure to cold, or exertion, and leading to rapid loss of power with the symptoms of an acute ascending paralysis. (*b*) As a sequel of the infectious diseases, such as small-pox, typhus, and measles. (*c*) As a result of traumatism, either fracture of the spine or very severe muscular effort. Concussion without fracture may produce it, but this is rare. Acute myelitis, for instance, scarcely ever follows railway accidents. (*d*) In diseases of the bones of the spine, either caries or cancer. This is a more common cause of localized acute transverse myelitis than of the diffuse affection. (*e*) In disease of the cord itself, such as tumors and syphilis; in the latter, either in association with gummata, in which case it is usually a late manifestation; or it may follow within a year or eighteen months of the primary affection.

Morbid Anatomy.—In localized acute myelitis affecting white and gray matter, as met with after accident or an acute compression, the cord is swollen, the pia injected, the consistence greatly reduced, and on incising the membrane an almost diffluent fluid may escape. In less intense grades, on section at the affected area, the distinction between the gray and white matter is lost, or is extremely indistinct. The tissue may be injected, or, as is often the case, hæmorrhagic. It is particularly in these forms, due to extension of disease from without or to acute compression, that we find definite involvement of the white matter. In other instances the gray matter is chiefly affected. There may be localized areas throughout the cord in which the gray matter is reduced in consistence and hæmorrhagic, the so-called red softening. There may be definite cavity formations in these foci. In some cases of disseminated or focal myelitis the meninges also are involved and there is a myelomeningitis. And, lastly, there are instances in which, throughout a long section of the cord, sometimes through the lumbar and the greater part of the thoracic, or in the thoracic and cervical regions, there is a diffuse myelitis of the gray substance.

Histologically the nerve fibres are much swollen and irregularly distorted, the axis cylinders are beaded, the myelin droplets are abundant, and the laminated bodies known as corpora amylacea may be seen. The granular fatty cells are also numerous and there may be leucocytes and red blood-corpuscles. Changes in the blood-vessels are striking; the smaller veins are distended and may show varicosities. The perivascular lymph spaces contain numerous leucocytes, and the smaller arteries themselves are frequently the seat of hyaline thrombi. The ganglion cells are swollen and irregular in outline, the protoplasm is extremely granular and vacuolated, and the nuclei, though usually invisible, may show signs of division, and the processes of the cells are not seen.

In cases which persist for some time we have an opportunity of seeing the later stages of acute myelitis. The acute, inflammatory, hyperæmic or red softening is succeeded by stages in which the affected area becomes more yellow from gradual alteration of the blood-pigment, and finally white in color from the advancing fatty degeneration. In cases of compression myelitis, a sclerosis may gradually be produced with the anatomical picture of a chronic diffuse myelitis.

Symptoms.—(*a*) *Acute Central Myelitis.*—~~It is~~ this form which ~~comes on spontaneously after~~ cold, or in connection with syphilis or one of the infectious diseases, or is seen in a typical manner in the extension from injuries or from tumor. The onset, though scarcely so abrupt as in hæmorrhage, may be sudden; a person may be attacked on the street and have difficulty in getting home. In some instances, the onset is preceded by pains in the legs or back, or a girdle sensation is present. It may be marked by chills, occasionally by convulsions; fever is usually present from the beginning—at first slight, but subsequently it may become high.

The *motor* functions are rapidly lost, sometimes as quickly as in Landry's ascending paralysis. The paraplegia may be complete, and, if the myelitis extends to the cervical region, there may be impairment of motion, and ultimately complete loss of power of the upper extremities as well. The sensation is lost, but there may at first be hyperæsthesia. The reflexes in the initial stage are increased, but in acute central myelitis, unless limited in extent to the thoracic and cervical regions, the reflexes are usually abolished. The rectum and bladder are paralyzed. Trophic disturbances are marked; the muscles waste rapidly; the skin is often congested, and there may be localized sweating. The temperature of the affected limbs may be lowered. Acute bed-sores may develop over the sacrum or on the heels, and sometimes a multiple arthritis is present. In these acute cases the general symptoms become greatly aggravated, the pulse is rapid, the tongue becomes dry; there is delirium, the fever increases, and may reach 107° or 108°.

The course of the disease is variable. In very acute cases death follows in from five to ten days. The cases following the infectious diseases, particularly the fevers and sometimes syphilis, may run a milder course.

The *diagnosis* of this variety of acute myelitis is rarely difficult. In common with the acute ascending paralysis of Landry, and with certain cases of multiple neuritis, it presents a rapid and progressive motor paraly-

sis. From the former it is distinguished by the more marked involvement of sensation, the trophic disturbances, the paralysis of bladder and rectum, the rapid wasting, the electrical changes, and the fever. From acute cases of multiple neuritis it may be more difficult to distinguish, as the sensory features in these cases may be marked, though there is rarely, if ever, in multiple neuritis complete anæsthesia; the wasting, moreover, is more rapid in myelitis. The bladder and rectum are rarely involved—though in exceptional cases they may be—and, most important of all, the trophic changes, the development of bullæ, bed-sores, etc., are not seen in multiple neuritis.

(*b*) *Acute Transverse Myelitis.*—The symptoms naturally differ with the situation of the lesion.

(1) Acute transverse myelitis in the *thoracic region*, the most common situation, produces a very characteristic picture. The symptoms of onset are variable. There may be initial pains or numbness and tingling in the legs. The paralysis may set in quickly and become complete within a few days; but more commonly it is preceded for a day or two by sensations of pain, heaviness, and dragging in the legs. The paralysis of the lower limbs is usually complete, and if at the level, say, of the sixth thoracic vertebra, the abdominal muscles are involved. Sensation may be partially or completely lost. At the onset there may be numbness, tingling, or even hyperæsthesia in the legs. At the level of the lesion there is often a zone of hyperæsthesia, which is discovered by passing a test-tube containing hot water along the spine, when the sensation of warmth changes to one of actual pain. A girdle sensation may occur early, and when the lesion is in this situation it is usually felt between the ensiform and umbilical regions. The reflex functions are variable. There may at first be abolition of the reflexes; subsequently, the reflexes, which pass through the segments lower than the one affected, may be exaggerated and the limbs may take on a condition of spastic rigidity. It does not always happen, however, that the reflexes are increased in a total transverse lesion of the cord. They may be entirely lost, as first pointed out by Bastian. That this is not due to the preliminary shock is shown by the fact that the abolition of the reflexes may continue for four or more months. The trophic changes are not marked. The muscles become extremely flabby, but not wasted in an extreme degree; subsequently rigidity develops. If the gray matter of the lumbar cord is involved, the flaccidity persists and the wasting may be considerable. The reaction of degeneration is not present. The temperature of the paralyzed limbs is variable. It may at first rise, then fall and become subnormal. Lesions of the skin are not uncommon, and bed-sores are apt to form. There is at first retention of urine and subsequent incontinence. If the lumbar centres are involved, there are from the outset vesical symptoms. The urine is alkaline in reaction and may rapidly become ammoniacal. The bowels are constipated and there is usually incontinence of the fæces. Some writers attribute the cystitis associated with transverse myelitis to disturbed trophic influence.

The course of complete transverse myelitis depends a good deal upon its cause. Death may result from extension. Segments of the cord may

be completely and permanently destroyed, in which case there is persistent paraplegia. The pyramidal fibres below the lesion undergo the secondary degeneration, and there is an ascending degeneration of the dorsal median columns. If the lower segments of the cord are involved the legs may remain flaccid. In some instances a transverse myelitis of the thoracic region involves the ventral horns above and below the lesion, producing flaccidity of the muscles, with wasting, fibrillar contractions, and the reaction of degeneration. More commonly, however, in the cases which last many months there is more or less rigidity of the muscles with spasm or persistent contraction of the flexors of the knee.

(2) *Transverse Myelitis of the Cervical Region.*—If the lesion is at the level of the sixth or seventh cervical nerves, there is paralysis of the upper extremities, more or less complete, sometimes sparing the muscles of the shoulder. Gradually there is loss of sensation. The paralysis is usually complete below the point of lesion, but there are rare instances in which the arms only are affected, the so-called cervical paraplegia. In addition to the symptoms already mentioned there are several which are more characteristic of transverse myelitis in the cervical region, such as the occurrence of vomiting, hiccough, and slow pulse, which may sink to 20 or 30, pupillary changes—myosis—sometimes attacks of dysphagia, dyspnœa, or syncope.

Treatment of Acute Myelitis.—In the rapidly developing form due either to a diffuse inflammation in the gray matter or to transverse myelitis, the important measures are: Scrupulous cleanliness, care and watchfulness in guarding against bed-sores, the avoidance of cystitis, either by systematic catheterization or, if there is incontinence, by a carefully adjusted bed urinal, or the use of antiseptic cotton-wool repeatedly changed. In an acute onset in a healthy subject the spine may be cupped. Counter-irritation is of doubtful advantage. Chapman's ice-bag is sometimes useful. No drugs have the slightest influence upon an acute myelitis, and even in subjects with well-marked syphilis neither mercury nor iodide of potassium is curative. Tonic remedies, such as quinine, arsenic, and strychnia, may be used in the later stages. When the muscles have wasted, massage is beneficial in maintaining their nutrition. Electricity should not be used in the early stages of myelitis. It is of no value in the transverse myelitis in the thoracic region with retention of the nutrition in the muscles of the leg.

V. DIFFUSE AND FOCAL DISEASES OF THE BRAIN.

I. TOPICAL DIAGNOSIS.

Only certain regions of the brain give localizing symptoms. These are the cortical motor centres, the speech centres, the centres for the special senses, and the tracts which connect these cortical areas with each other and with other parts of the nervous system.

The following is a brief summary of the effects of lesions from the cortex to the spinal cord:

1. **The Cerebral Cortex.**—(*a*) Destructive lesions of the motor cortex (central gyri, lobulus paracentralis, posterior portions of the three frontal gyri, especially of the inferior) cause *spastic paralysis* in the muscles of the opposite side of the body. The paralysis is at first flaccid, but contractures subsequently develop. The extent of the paralysis depends upon that of the lesion. It is apt to be limited to the muscles of the face or of an extremity, giving rise to the cerebral monoplegias (Fig. 11, 1). One group of muscles may be much more affected than others, especially in lesions of the highly differentiated area for the upper extremity. It is uncommon to find all the muscle groups of an extremity equally involved in cortical monoplegia. Very rarely through small bilaterally symmetrical lesions monoplegia of the tongue may result without paralysis of the face. A lesion may involve centres lying close together or overlapping one another, thus producing associated monoplegias—e. g., paralysis of the face and arm, or of the arm and leg, but not of the face and leg without involvement of the arm. Very rarely the whole motor cortex is involved, causing paralysis of the opposite side—cortical hemiplegia. Usually in such instances there is marked recovery, so that only a monoplegia persists.

The motor area corresponds also, at least in large part, to the region of the cortex in which the impulses concerned in general bodily sensation (cutaneous sensibility, muscle sense, visceral sensations) first arrive (the somæsthetic area). Combined with the muscular weakness there is usually some disturbance of sensations, particularly of those of the muscular sense. The stereognostic sense is very often affected. In brachial monoplegia, for example, a coin or a knife when placed in the hand of the paralyzed limb, the patient's eyes being closed, is not recognized, owing to inappreciation of the form and consistence of the object, and this even though the slightest tactile stimulus applied to the fingers or surface of the hand is felt and may be correctly localized. The sense of touch, pain, and temperature may be lowered, but usually not markedly unless the superior and inferior parietal lobules are involved in addition to the central gyri. Paræsthesias and vaso-motor disturbances are common accompaniments of paralyses of cortical origin.

(*b*) Irritative lesions cause localized spasms as described above. The most varied muscle groups corresponding to particular movement forms may be picked out. If the irritation be sudden and severe, typical attacks of Jacksonian epilepsy may occur. These convulsions are usually preceded and accompanied by subjective sensory impressions. Tingling or pain, or a sense of motion in the part, is often the *signal symptom* (Seguin), and is of great importance in determining the seat of the lesion. Here, too, the stereognostic sense is frequently involved.

Lesions are often both destructive and irritative, and we then have combinations of the symptoms produced by each. For instance, certain muscles may be paralyzed, and those represented near them in the cortex may be the seat of localized convulsions, or the paralyzed limb itself may be at times subject to convulsive spasms, or muscles which have been convulsed may become paralyzed. The close observation of the sequence of the symptoms in such cases often makes it possible to trace the progress

of a lesion involving the motor cortex. In these cases the most frequent cause is a developing tumor, though sometimes local thickenings of the membranes of the brain, small abscesses, minute hæmorrhages, or fragments of a fractured skull must be held responsible.

In another section lesions involving the centres for the special senses are considered, and we shall simply refer to them here. The symptoms caused by lesions of the speech centres will be described under aphasia, and it is only necessary to note here the near situation of the motor speech area (Broca's centre) in the left inferior frontal convolution to the centres for the face and arm on that side, and to state that motor aphasia is often associated with monoplegia of the right side of the face and the right arm. Accompanying the paralysis, following a Jacksonian fit, of the right face or arm there is often a transient motor aphasia.

According to Flechsig, the sensori-motor centres are limited to tolerably circumscribed areas in the cortex, which differ from other portions in that they are provided with projection fibres which connect them with lower centres. The remaining areas of the cortex, amounting, he believes, to about two thirds of the whole, are devoid of projection fibres and are concerned entirely in associative activities. These latter areas, the "association centres" of Flechsig, are three in number: (1) The anterior association centre, including the whole of the frontal lobe in front of the somæsthetic area; (2) the middle association centre, corresponding to the cortex of the island of Reil; and (3) the large, posterior association centre, including the præcuneus, the superior and inferior parietal lobules, the supramarginal and angular gyri, and the whole of the temporal and occipital lobes except the auditory and visual sensory areas.

Flechsig attributes the higher psychic functions, especially those connected with the personality of the individual, to the anterior association centres, while the intellectual activities which have to do with knowledge of the external world he believes correspond to the functions of the large posterior association centre. Whether these views be true, and, if so, in how far they may be applied practically in the localization of diseases, especially of the mind, the future has to decide.

2. **Centrum Semiovale.**—Lesions in this part may involve either projection fibres (motor or sensory) or association fibres. If involvement of the motor path cause paralysis, this has the distribution of a cortical palsy when the lesion is near the cortex, and of a paralysis due to a lesion of the internal capsule when it is near that region. These lesions of the motor fibres may be associated with symptoms due to interruption in the other systems of fibres running in the centrum semiovale; there may be sensory disturbances—hemianæsthesia and hemianopia—and if the lesion is in the left hemisphere one of the different forms of aphasia may accompany the paralysis.

3. **Corpus Callosum.**—This may be congenitally absent without symptoms. An acute lesion involving a large portion of the corpus callosum may, however, yield symptoms suggestive of its localization in this region. In the case recorded by Reinhard, in which the situation of the lesion was suspected ante-mortem, there was disturbance of equilibration (without

vertigo) and of the synergetic movements of both halves of the body. The autopsy revealed a gliosarcoma which had destroyed the posterior three fourths of the corpus callosum. In Bristowe's 4 cases there existed, as symptoms common to all, pain in the head and partial or complete hemiplegia, with gradual extension of the paralysis to the opposite side of the body. Toward the end of life there was disturbance of speech, difficulty in deglutition, incontinence of urine and fæces and dementia. Here the symptoms have in them nothing that can be looked upon as pathognomonic; indeed, many of the phenomena were doubtless dependent upon involvement of the projection and association fibres of the centrum semiovale.

In animals in which the corpus callosum has been cut experimentally progressive emaciation has been mentioned as a characteristic phenomenon.

4. Internal Capsule (Fig. 4).—Through this pass within a rather narrow area all, or nearly all, of the projection fibres (both motor and sensory) which are connected with the cerebral cortex. It is divided into an anterior limb, a knee, and a posterior limb, the latter consisting of a thalamo-lenticular portion (its anterior two thirds) and a retro-lenticular portion (its posterior third). In considering the effects of a given focal lesion involving the fibres of the internal capsule, it is not to be forgotten that the relations of the two limbs of the capsule to one another and to the knee vary considerably in different horizontal planes. Much of the confusion in the bibliography is dependent upon neglect to describe the horizontal level of the lesion, as well as its situation in an antero-posterior direction. The principal bundle passing through the anterior limb of the capsule is that which connects the frontal gyri and the medial bundle in the base of the peduncle (crus) with the nuclei of the pons. These fibres are centrifugal, and innervate chiefly the lower motor nuclei governing bilaterally innervated muscles, especially those of the eyes, head, neck, and probably those of the mouth, tongue, and larynx. In lower horizontal planes these fibres are situated near the knee of the capsule. It is the region of the knee of the capsule which transmits especially the fibres passing from the cerebral cortex to the nuclei of the facial, hypoglossal and third nerves. The path which supplies the nuclei governing the muscles used in speech passes through the knee.

The pyramidal tract goes through the thalamo-lenticular portion of the capsule. The motor fibres are arranged according to definite muscle groups, or rather movement forms, those for the movements of the arm being anterior to those for the leg. The number of fibres for a given muscle group corresponds rather to the degree of complexity of the movements than to the size of the muscles concerned. Thus the areas for the fingers and toes are relatively large.

The fibres to the somæsthetic area of the cortex—that is, those from the ventro-lateral group of nuclei of the thalamus and the tegmental radiations—carrying impulses concerned in general bodily sensation, pass upward through the posterior part of the thalamo-lenticular portion of the capsule. Some of these fibres pass through the anterior two thirds of the posterior limb alongside of the fibres of the pyramidal tract.

Through the retro-lenticular portion of the posterior limb, opposite the

posterior third of the lateral surface of the thalamus, pass (1) the fibres carrying impulses concerned in the sensations of the opposite visual field (optic radiation from the lateral geniculate body to the visual sense area in the occipital cortex); (2) the fibres carrying impulses concerned in auditory sensations (radiation from the medial geniculate body to the auditory sense area in the cortex of the temporal lobe); (3) the fibres (probably centrifugal) connecting the cortex of the temporal lobe with the nuclei of the pons.

With this preliminary knowledge concerning the internal capsule, it is not difficult to understand the symptoms which result when it is diseased.

Since here all the fibres of the upper motor segment are gathered together in a compact bundle, a lesion in this region is apt to cause complete hemiplegia of the opposite side, followed later by contractures; and if the lesion involves the hinder portion of the posterior limb there is also hemianæsthesia, including even the special senses (Fig. 4). As a rule, however, lesions of the internal capsule do not involve the whole structure. The disease usually affects mainly either the anterior or posterior portions, and even in instances in which at first the symptoms point to total involvement, there is a disappearance often of a large part of the phenomena after a short time. Thus when the pyramidal tract is destroyed (lesion of the thalamo-lenticular portion of the capsule) the arm may be affected more than the leg, or *vice versa*. The facial paralysis is usually slight, though if the lesion be well forward in the capsule the paralysis of the face and tongue may be marked.

Hemianæsthesia alone without involvement of the motor fibres, due to disease of the capsule, is rare. There is usually also at least partial paralysis of the leg. When the retro-lenticular portion of the capsule is destroyed the hemianæsthesia is accompanied by hemianopsia, disturbance of hearing, and sometimes of smell and taste. The occurrence of hemichorea, marked tremor, or hemiathetosis after a capsular hemiplegia points to the involvement of the thalamus or of the hypothalamic region in the lesion.

Charcot and others have described cases in which as a result of disease of the internal capsule there has been paralysis of the face and leg without involvement of the arm. In such instances the lesion is linear, extending from the posterior part of the anterior limb of the internal capsule backward and lateralward to the leg region in the posterior limb of the capsule, the region for the arm escaping.

Capsular lesions when pure are not accompanied by aphasic symptoms, alexia, or agraphia. A "subcortical" motor aphasia may occur, if along with complete destruction of the anterior limb of the internal capsule on one side there be associated a lesion of the caudate nucleus on the opposite side large enough to interfere with the adjacent fibres going to the nuclei governing the muscles of speech.

5. **Crura (Cerebral Peduncles).**—From this level through the pons, medulla, and cord the upper and lower motor segments are represented, the first by the fibres of the pyramidal tracts and by the fibres which go from the cerebral cortex to the nuclei of the cerebral nerves, the latter by the motor nuclei and the nerve fibres arising from them. Lesions often affect

both motor segments, and produce paralyses having the characteristics of each. Thus a single lesion may involve the pyramidal tract and cause a spastic paralysis on the opposite side of the body, and also involve the nucleus or the fibres of one of the cerebral nerves, and so produce a lower segment paralysis on the same side as the lesion—crossed paralysis. In the crus the third and fourth cerebral nerves run near the pyramidal tract, and a lesion of this region is apt to involve them or their nuclei, causing partial paralysis of the muscles of the eye on the same side as the lesions, combined with a hemiplegia of the opposite side (Fig. 10, 3).

The optic tract also crosses the crus and may be involved, giving hemianopsia in the opposite halves of the visual fields.

If the tegmentum be the seat of a lesion which does not involve the base of the peduncle (or pes) there may be disturbances of cutaneous and muscular sensibility, ataxia, disturbances of hearing, or oculo-motor paralysis. An oculo-motor paralysis of one side, accompanied by a hemi-ataxia of the opposite side, appears to be especially characteristic of a tegmental lesion.

6. **Corpora Quadrigemina.**—Anatomical studies point to the view that the superior colliculus (anterior quadrigeminal body) represents the most important subcortical central organ for the control of the eye-muscle nuclei. This is supported to a certain extent by clinical evidence, though as yet but few cases have been carefully studied. Sight may be only slightly, if at all, disturbed when the superior colliculus is destroyed, and color vision may remain normal. The pupil is usually widened, and the pupillary reaction, both to light and on accommodation, interfered with. Apparently actual paralysis of the eye muscles does not occur unless the nucleus of the third nerve ventral to the aqueduct be also injured.

The inferior colliculus (posterior quadrigeminal body), on the other hand, has been shown by anatomical study to be an important way-station in the auditory conduction-path. A large part of the lateral lemniscus ends in its nucleus, and from it emerge medullated fibres which pass through the brachium quadrigeminum inferior to the medial geniculate body. Thence a large bundle runs through the retro-lenticular portion of the internal capsule to the auditory sense area in the cortex of the temporal lobe.

Weinland has collected 19 cases of tumors of the corpora quadrigemina from the bibliography; in 9 of these auditory disturbances were especially noted. Since the central auditory path of each side receives impulses from both ears, lesion of the colliculus on one side may dull the hearing on both sides, though the opposite ear is usually the more defective. Lesion of the inferior colliculus may be accompanied by disturbance of mastication, owing to paralysis of the descending (mesencephalic) root of the trigeminus. The fourth nerve may also be involved. The ataxia which sometimes accompanies lesions of the corpora quadrigemina is probably to be referred to disturbance in conduction in the medial lemniscus.

7. **Pons and Medulla Oblongata.**—Lesions involving the pyramidal tract, together with any one of the motor cerebral nerves of this region, cause crossed paralysis. A lesion in the lower part of the pons is apt to

cause a lower-segment paralysis of the face on the same side (destruction of the nucleus of the facial nerve or of its root fibres) and a spastic paralysis of the arm and leg on the opposite side (injury to pyramidal tract) (Fig. 10, 4). The abducens, the motor part of the trigeminus, and the hypoglossus nerves may also be paralyzed in the same manner. When the central fibres to the nucleus of the hypoglossus are involved a peculiar form of anarthria results. If the nucleus itself be diseased, swallowing is interfered with.

When the sensory fibres of the fifth nerve are interrupted, together with the sensory tract (the medial lemniscus or fillet) for the rest of the body, which has already crossed the middle line, there is a crossed sensory paralysis—i. e., disturbed sensation in the distribution of the fifth on the side of the lesion, and of all the rest of the body on the opposite side.

A paralysis of the external rectus muscle of one eye and of the internal rectus of the other eye (conjugate paralysis of the muscles which turn the eye to one side), in the absence of a "forced position" of the eyeballs, is highly characteristic of certain lesions of the pons. In such cases the internal rectus may still be capable of functioning on convergence, or when the eye to which it belongs is tested independently of that in which the external rectus is paralyzed. This form of paralysis is found, as a rule, only when the nodule lies just in front of the abducens or involves the nucleus itself, or includes, besides the root fibres of the abducens, that portion of the formatio reticularis that lies between them and the fasciculus longitudinalis medialis (von Monakow). The cases of conjugate paralysis just referred to may be complicated by other disturbances of the eye-muscle movements, in which case the interpretation of the symptoms may be rendered difficult. The facial nerve is often involved in these paralyses.

In lesions of the pons the patient often has a tendency to fall toward the side on which the lesion is, probably on account of implication of the middle peduncle of the cerebellum (brachium pontis). Still more frequent is the simple motor hemi-ataxia consequent upon lesion of the medial lemniscus, and perhaps of longitudinal bundles in the formatio reticularis. This is often accompanied by disturbance of muscular and cutaneous sensations. Only when the lesion is very extensive are there disturbances of hearing (involvement of the lateral lemniscus or corpus trapezoideum).

The symptoms produced by involvement of the different cerebral nerves will be considered in detail in another section.

8. **Cerebellum.**—The functions of this part of the brain are still under consideration. Luciani, whose monograph is exhaustive, regards it as "an end organ, directly or indirectly related to certain peripheral sensory organs and in direct efferent relationship with certain ganglia of the cerebro-spinal axis, and indirectly with the motor apparatus in general. It is functionally homogeneous, each part exercising the functions of the whole, but having special relations to the muscles of the corresponding side of the body" (Krauss).

Lesions of the lateral lobes affect the corresponding side of the body, while lesions of the middle lobe (vermis) affect both sides. Partial removal

is followed by transient muscular weakness; complete removal by extreme incoördination. Its one important function would appear to be the coördination of the muscular movements.

In monkeys the symptoms differ much at different periods after the operation. During the first five or six days irritation phenomena predominate. There is, according to Luciani, asthenia, atony, and astasia of the muscles on the side of the body operated upon. The animal cannot stand or walk. All these symptoms may gradually disappear in the course of a few months.

W. C. Krauss has analyzed the lesions and symptoms in 100 cases of disease of this part. The morbid conditions were as follows: Sarcoma in 22 cases; tubercle in 22; glioma in 18; abscess in 10; tumor of unspecified origin in 13; cyst in 7; and 1 case each of softening, endothelioma, cyst and sarcoma, cancer, gumma, fibroma, and hæmorrhage. The left lobe was affected 32 times, the right lobe 32 times, and the middle lobe 17 times. Thus tumor constituted by far the most important affection. There may be no symptoms whatever if it is in one hemisphere only and does not involve the middle lobe. There are not only instances of complete absence of one whole hemisphere, but also of extensive bilateral disease which throughout life have yielded no noticeable symptoms. Other portions of the brain appear to be able to take on the functions normally performed by the cerebellum.

The experiments of J. S. Risien Russell do not entirely confirm the observations of Luciani. In the first place, the occurrence of asthenia is not constant, and as to atony, while the patellar tendon reflexes are sometimes absent, they are as a rule intact in pure cerebellar lesions. There may be even muscular rigidity instead of atony. Russell's experiments make it seem likely that the cerebellar hemisphere of one side exercises constantly an inhibitory effect upon the activities of the cerebral hemisphere of the opposite side (probably by way of the brachium conjunctivum). Thus after removal of one cerebellar hemisphere he found that much milder faradic stimulation of the contra-lateral motor area would call forth movements of the arm and leg than that necessary to stimulate the homo-lateral motor area. The epileptic seizures following the administration of absinthe were far greater on the side of ablation. It is not impossible that the explanation of the epileptiform attacks by no means rare in cerebellar disease is here to be sought. The most common symptoms in tumor of the cerebellum are as follows:

Vertigo, which is more constant in this than in affections of any other region of the brain. Some believe this to be due to involvement of the nervus vestibularis or its nuclei of termination, by means of which the semicircular canals are connected with the cerebellum. The symptom was present in 48 of the cases of Krauss's collection, not reported in 43. The vertigo appears to be entirely independent of the ataxia. Though most frequently associated, either symptom may be present without the other. The vertigo of cerebellar disease is often associated with the feeling that objects are revolving about the body, or that the body itself is moving. *Headache* was present in 83 cases. *Vomiting* occurred in 69 cases, not re-

ported in 23. *Optic neuritis* was found in 66 cases, not reported in 23. Very serious disturbances of vision may result from pressure on the aqueductus cerebri, leading to increased pressure in the third ventricle; this, through bulging of the floor, can directly injure the chiasm or optic nerve.

Of symptoms which are designated as more particularly cerebellar, *ataxia* is the most important. In cerebellar ataxia the gait is irregular and staggering, often zigzag, and in attempting to walk the patient sways to and fro like a drunken man (*démarche d'ivresse* of the French writers). As a rule, the patient walks and tends to fall toward the affected side, but the rule is not certain. The ataxia of cerebellar disease is to be sharply differentiated from the ataxia of tabes dorsalis, from cortical ataxia, and probably from the ataxia accompanying diseases of the tegmental portion of the pons and cerebral peduncle. Cerebellar ataxia is both static and dynamic. The opening or closing of the eyes is of less influence than in spinal ataxia. Very important for differential diagnosis is the fact that when the patient lies in bed movements tolerably well coördinated can be carried out. The coarse nature of the incoördination distinguishes cerebellar ataxia from that due to lesion of the cerebral cortex. In the latter the finer movements (buttoning, etc.) are especially apt to be involved, and there is usually hemi-paresis or mono-paresis, and often disturbance of muscular sense and of the stereognostic sense (von Monakow). Cerebellar ataxia may depend upon the withdrawal of the influence of the cerebellum upon the cerebrum.

Paresis of the trunk muscles, manifest in an inability to perform the movements of bending, erection, and lateral flexion of the trunk, may be present (Hughlings Jackson). Risien Russell holds that the paralysis is "probably directly due to the withdrawal of the cerebellar influence from the muscles."

Other less constant but suggestive symptoms are neuralgic pains in the region of the neck and occiput; blocking of the venæ Galeni and dilatation of the lateral ventricles, causing in children hydrocephalus; pressure on the mid-brain, pons, or medulla oblongata, producing paralysis of the cerebral nerves, rhythmical contractions of the head or extremities, nystagmus, tremor, anarthria, auditory or visual disturbances. There may be glycosuria and bilateral rigidity from pressure on the motor paths. Sudden death may occur. Forced movements, especially rotation of the trunk, forced positions (of the head or trunk), and a peculiar forced position of the eyes (one turned downward and to the side, the other upward and inward) are almost pathognomonic of disease of one brachium pontis (middle cerebellar peduncle).

The reflexes are very variable; they were absent in 12 cases. In pure cerebellar lesion they are probably intact or exaggerated, but when the cerebellar disease involves other structures, directly or indirectly, through action at a distance, or when there is associated disease of the spinal tracts, the reflexes may be abolished.

Symptoms of general mental disturbance may accompany cerebellar disease, but they are not characteristic. There is often irritability, enfeebled memory, and toward the end sopor and coma.

II. APHASIA.*

The speech mechanism consists of receptive, perceptive, and emissive centres in the cortex cerebri, disturbances of which cause *aphasia*, and centres in the medulla which preside over the muscles of articulation, disturbances of which produce *anarthria*, the condition of gradual loss of power of speech, such as occurs in bulbar paralysis. To the disturbances of speech resulting from lesion of the white fibres throwing the lower nuclei governing the speech muscles under the influence of the cortex, without primary injury to either the cortex of the nuclei in the medulla, the term *aphemia* has been applied (Bastian).

The studies of Dax, Broca, Bastian, Kussmaul, Wernicke, Lichtheim, and others have widened enormously our knowledge of speech disorders. Language is gradually acquired by imitation. During development in order that we may make ourselves understood (expressive components of speech), it is necessary that we learn to understand the expressions of others (perceptive speech components). Thus, in teaching a child to say *bell*, the sound of the uttered word enters the afferent path (auditory nerve) and reaches the auditory perceptive centre, from which an impulse is sent to the emissive or motor centre presiding over the nuclei in the medulla, through which the muscles of articulation are set in action. The arc in Lichtheim's schema (Fig. 9) is *a* A, M *m*. The child gradually acquires in this way *memories of the sounds of words*, which are stored at the centre A, and *motor memories*—the kinæsthetic memories of the coördinated muscular movements of the lips, tongue, and larynx necessary to utter words—which are stored at the centre M (glosso-kinæsthetic centre of Bastian). In a similar manner, when shown the bell, the child acquires *visual memories*, which are conveyed through the optic nerve to the visual perceptive centres, *o* O. So also with the memories of the sound of the bell when struck. The memory picture of the shape of the bell, the memory of the appearance of the word bell as written or printed, and the motor memories of the muscular movements required to write the word are distinct from each other; yet they are intimately connected, and form together what is termed the *word-image*. In addition to all this the child gradually acquires in his education ideas as to the use of the bell—intellectual conceptions—the centre for which is represented at I in the diagram. In volitional or intellectual speech, as in uttering the word *bell*, the path would be I, M *m*, and in writing the word, I, M, W, *h*. These various "memories" are as a rule stored or centred in the left hemisphere (see Fig. 3). When the word "bell" is heard, the mental state which results includes not only the activities of the auditory perception-centre, but also by association the activities

* A large number of valuable works on aphasia have appeared within the past few years, chief of which may be placed Bastian's recently issued monograph (1898). The works of Wyllie and Elder and the lectures of Bramwell (British Medical Journal, 1897–'98), the monograph of Collins, the text-book of C. K. Mills, and the various publications of Eskridge, von Monakow's volume in Nothnagel's Handbuch, and Miraillée's work are among the most important recent contributions.

of a whole series of cerebral centres, which in the manifold experiences of life have been occupied at one time or another in some way with some psychic attribute of the external object, or with combining and coördinating various impressions of it.

The relations of language (heard, read, spoken, and written) involves then (*a*) sensory perceptive centres (hearing and sight and, in the blind,

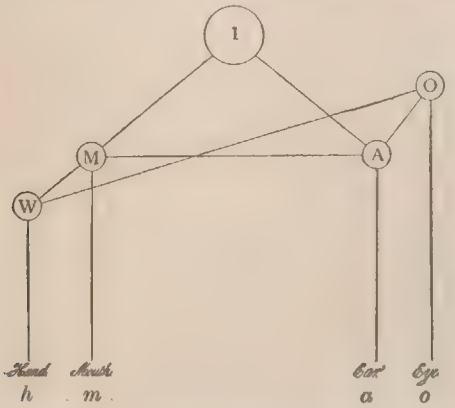

FIG. 9.—Lichtheim's schema. A, auditory area in cerebral cortex, in which are stored the memories of the sounds of words; *a* A, auditory conduction path from cochlea to temporal lobe; O, visual area in cerebral cortex, perception centre for written and printed words; *o* O, visual conduction path from retina to occipital lobe; M, speech centre in which are stored the memories of the muscular movements which produce spoken words (Bastian's glosso-kinæsthetic centre); M *m*, path along which impulses travel to innervate the lower nuclei which govern the muscles concerned in speech; W, area in cerebral cortex in which are stored the memories of the muscular movements concerned in writing (Bastian's cheiro-kinæsthetic centre); W *h*, path along which impulses travel to innervate the lower nuclei which govern the muscles used in writing; I, areas of association in cortex by means of which the activities of the various sensory perception centres may be united to higher units (conceptions, ideas, thoughts, etc.), and whence the centres M and W may be incited.

touch); (*b*) emissive or motor centres for speech and writing; and (*c*) higher psychical centres, through which we obtain an intellectual conception of what is said or written, and by which we express voluntarily our ideas in language.

Aphasic disturbances for convenience of description are arbitrarily divided into two chief forms—*sensory* and *motor*.

(1) **Sensory Aphasia; Apraxia; Word-blindness; Word-deafness.**—By apraxia is understood a condition in which there is loss or impairment of the power to recognize the nature and characteristics of objects. Persons so affected act "as if they no longer possessed such object memories, for they fail to recognize things formerly familiar. A fork, a cane, a pin, may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term blindness of mind was used, is found to extend to other senses than that of sight. Thus the tick of a watch, the sound of a bell, a melody of music, may fail to arouse the idea which it

formerly awakened, and the patient has then deafness of mind; or an odor or taste no longer calls up the notion of the thing smelled or tasted; and thus it is found that each or all of the sensory organs, when called into play, may fail to arouse an intelligent perception of the object exciting them. For the general symptoms of inability to recognize the use or import of an object the term *apraxia* is now employed" (Starr).

Apraxia may occur alone, but more commonly is associated with varieties of sensory and motor aphasia. The patient may be able to read, but the words arouse no intelligent impression in his mind. While blind to memory-pictures aroused through sight, the perceptions may be stimulated by touch; thus there are instances on record of apraxic patients unable to read by sight, who could on tracing the letters by touch name them correctly. Of the forms of apraxia, mind-blindness and mind-deafness are the most important.

The cases of *mind-blindness* collected by Starr indicate that the lesion exists in the left hemisphere in right-handed persons, and in the right hemisphere in left-handed persons. The disease usually involves the angular and supramarginal gyri or the white matter beneath them. Blindness of the "mind's eye" may at times be functional and transitory, and is associated with many forms of mental disturbance. In a remarkable case reported by Macewen, the patient, after an injury to the head, had suffered with headache and melancholia, but there was no paralysis. He was psychically blind and though he could see everything perfectly well and could read letters, objects conveyed no intelligent impression. A man before his eyes was recognized as some object, but not as a man until the sounds of the voice led to the recognition through the auditory centres. The skull was trephined over the angular gyrus and the inner table was found to be depressed and a portion had been driven into the brain in this region. The patient recovered. Mind-blindness is the equivalent of visual amnesia. Other manifestations of mind-blindness are met with; thus a young man with secondary syphilis had several convulsive seizures, after one of which he remained unconscious for some time. On awakening, the memory-pictures of faces and places were a blank, and he neither knew his parents nor brothers, nor the streets of the town in which he lived; he had no aphasia proper, and no paralysis. Again, there may be complete tactile amnesia, as in the cases reported by C. W. Burr.

Word-blindness may occur alone or with motor aphasia. In uncomplicated cases the patient is no longer able to recall the appearances of words, and does not recognize them on a printed or written page. The patient may be able to pronounce the letters and can often write correctly, but he cannot read understandingly what he has written. It is rare, however, for the patient to be able to write with any degree of facility. There are instances in which the patient, unable to read, has yet been able to do mathematical problems and to recognize playing cards. The lesions in cases of word-blindness is, in a majority of cases, in the angular and supramarginal gyri on the left side. It is commonly associated with hemianopia, and not infrequently with mind-blindness (Fig. 3).

Mind-deafness is a condition in which sounds, though heard and per-

ceived as such, awaken no intelligent perceptions. A person who knows nothing of French has mind-deafness so far as the French language is concerned, and though he recognizes the words as words when spoken, and can repeat them, they awaken no auditory memories. The musical faculties may be lost in aphasics, who may become note-deaf and unable to appreciate melodies or to read music (*amusia*). This may occur without the existence of motor aphasia, and, on the other hand, there are cases on record in which with motor aphasia for ordinary speech the patient could sing and follow tunes correctly. Mind-deafness is also known as auditory amnesia.

Word-deafness is a condition in which the patient no longer understands spoken language. The memory of the sound of the word is lost, and can neither be recalled nor recognized when heard. It is usually associated with other varieties of aphasia, though there are cases in which the patient has been able to read and write and speak. The lesion in word-deafness has been accurately defined in a number of cases to be in the posterior portion of the superior temporal convolution and the transverse temporal gyri on the left side (Fig. 3).

In *ordinary sensory aphasia* of Wernicke's type there is loss of power to understand spoken words and to repeat words pronounced before the patient. The patient, as a rule, cannot read (alexia), and is usually unable to express his thoughts in writing (agraphia). Spontaneous speech may be somewhat interfered with, and on account of the interference with speech control, resulting from the loss of memory of the sounds of words, there may be a little paraphasia.

In the so-called *pure word-deafness* (Wernicke's subcortical sensory aphasia) the symptoms differ from those of the most common form of sensory aphasia in that the power to read and to write are retained. Besides, there is but little if any paraphasia.

In the so-called *transcortical sensory aphasia* the patient has lost the power of understanding spoken words, although he is capable of spontaneous speech and also of repeating words pronounced before him. Spontaneous writing is impossible. He can read aloud from a manuscript or printed page, but does not understand what he reads. There is some paraphasia.

(2) **Ordinary motor or ataxic aphasia** is a condition in which the memory of the efforts necessary to pronounce words is lost, owing to disturbance in the emissive centres. This is the variety long ago recognized by Broca, the lesion of which was localized by him in the left inferior frontal convolution. The patient may not be able to utter a single word; more commonly he can say one or two words, such as "no," "yes," and he not infrequently is able to repeat words. When shown an object, though not able to name it, he may evidently recognize what it is. If told the name, he is, as a rule, unable to repeat it. A man knowing the French and German languages may lose the power of expressing his thoughts in them, while retaining his mother-tongue; or, if completely aphasic, may recover one before the other. As the third left frontal convolution is in close contact with the centres for the face and arm, these are not uncommonly involved, with the production of a partial or, in some instances, a complete right-

sided hemiplegia. *Alexia*, or inability to read, occurs with motor aphasia and also with word-blindness.

As a rule, in motor aphasia there is also inability to write—*agraphia*. When there is right brachial monoplegia it is difficult to test the capability, but there are instances of motor aphasia without paralysis, in which the power of voluntary writing is lost. The condition varies very much; thus a patient may not be able to write voluntarily or from dictation, and yet may copy perfectly. It is still a question whether there is a special writing centre. It has been placed by some writers at the base of the second frontal convolution, but it seems likely that it coincides with the motor area for the upper extremity. From the above type, which may be looked upon as the ordinary form of motor aphasia, two other varieties must be separated—viz., (1) pure word-dumbness and (2) the so-called transcortical motor aphasia.

Pure word-dumbness (subcortical motor aphasia of Lichtheim and Wernicke) is the term applied to that complex of symptoms occasionally met with, in which, though the power of spontaneous speech and of repeating words heard is lost, the individual can write, and can read to himself with understanding that which is written or printed. He is, of course, unable to read aloud.

Transcortical motor aphasia is the term applied by Wernicke to that form of motor aphasia studied first by Lichtheim in which the power to speak and write spontaneously is lost, though the patient can understand spoken and written words perfectly, can read aloud, can write to dictation, and can copy another individual's writing.

There is a form known as *mixed aphasia*, in which the patient understands what is said, and speaks even long sentences correctly, but he constantly tends to misplace words, and does not express his ideas in the proper words. It is precisely these cases which afford the most exquisite examples of paraphasia. All grades of this may be met with, from a state in which only a word or two is misplaced to an extreme condition in which the patient talks jargon. In these cases the association tract is interrupted between the auditory perceptive and the emissive centres, hence it is sometimes known as Wernicke's *aphasia of conduction*. The lesion is usually in the insula and in the convolutions which unite the frontal and temporal lobes. Lichtheim's schema, though out of accord with a number of facts, is extremely useful to the beginner, and will assist the student in obtaining a rational idea of the varieties of aphasia:

1. In the condition of apraxia or mind-blindness the ideation centres, I, are involved, often with the auditory and visual perceptive centres, A and O.

2. A lesion at A, the centre for the auditory memories of words (left superior temporal gyrus), is associated with word-deafness.

3. A lesion at O, the centre for visual memories (occipital cortex), causes word-blindness.

4. Interruption of the tracts uniting A M and O M causes the conduction aphasia of Wernicke—*paraphasia*.

5. Destruction of the centre M (Broca's convolution) causes ordinary motor aphasia, in which the patient cannot express thoughts in speech.

A lesion at M usually destroys also the power of writing. The centre for memories of the movements made in writing, W, is distinct from that of speech. It is called by Bastian the "cheiro-kinæsthetic" centre. A lesion at M, which would destroy the power of voluntary speech, might leave open the connections between O W and A W, by which the patient could copy or write from dictation. According to Wernicke's conception, pure word-deafness (subcortical sensory aphasia) would be due to a lesion in the path *a* A, transcortical sensory aphasia to a lesion in the path A I, pure word-dumbness (subcortical motor aphasia) to a lesion in the path M *m*, and transcortical motor aphasia to a lesion in the path I M. While undoubtedly there are groups of cases separable clinically corresponding to these various types, still pathological examinations have already shown that the nomenclature is faulty and will not stand, though the number of cases thus far thoroughly studied at autopsy does not suffice for the construction of a complete classification on a pathological basis.

The problems of aphasia are in reality excessively complicated, and the student must not for a moment suppose that cases are as simple as diagrams indicate. A majority of them are very complex, but with patience the diagnosis of the different varieties can often be worked out.

The following tests should be applied in each case of aphasia after having determined the presence or absence of paralyses, and whether the patient is right-handed or left-handed: (1) The power of recognizing the nature, uses, and relations of objects—i. e., whether apraxia is present or not; (2) the power to recall the name of familiar objects seen, smelled, or tasted, or of a sound when heard, or of an object touched; (3) the power to understand spoken words; (4) the capability of understanding printed or written language; (5) the power of appreciating and understanding musical tunes; (6) the power of voluntary speech—in this it is to be noted particularly whether he misplaces words or not; (7) the power of reading aloud and of understanding what he reads; (8) the power to write voluntarily and of reading what he has written; (9) the power to copy; (10) the power to write at dictation; and (11) the power of repeating words.

The *medico-legal aspects* of aphasia are of great importance. No general principle can be laid down, but each case must be considered on its merits. Langdon, in reviewing the whole question, concludes: "Sanity established, any legal document should be recognized when it can be proved that the person making it can understand fully its nature by any receptive channel (viz., hearing, vision, or muscular sense), and can, in addition, express assent or dissent with certainty to proper witnesses, whether this expression be by spoken speech, written speech, or pantomime."

Prognosis and Treatment.—In young persons the outlook is good, and the power of speech is gradually restored apparently by the education of the centres on the opposite side of the brain. In adults the condition is less hopeful, particularly in the cases of complete motor aphasia with right hemiplegia. The patient may remain speechless, though capable of understanding everything, and attempts at re-education may be futile. Partial recovery may occur, and the patient may be able to talk, but misplaces words. In sensory aphasia the condition may be only transient, and the

different forms rarely persist alone without impairment of the powers of expression.

The education of an aphasic person requires the greatest care and patience, particularly if, as so often happens, he is emotional and irritable. It is best to begin by the use of detached letters, and advance, not too rapidly, to words of only one syllable. Children often make rapid progress, but in adults failure is only too frequent, even after the most painstaking efforts. In the cases of right hemiplegia with aphasia the patient may be taught to write with the left hand.

III. AFFECTIONS OF THE BLOOD-VESSELS.

1. Hyperæmia.

Congestion of the brain has in the past played an important part in cerebral pathology. Undoubtedly there are great variations in the amount of blood in the cerebral vessels; this is universally conceded, but how far these changes are associated with a definite group of symptoms is not quite so clear. The whole subject has recently been revised by R. Geigel, who rightly insists that the nutrition of the nerve-cells and the possibility of interchange of gases between the blood and the cerebral tissues is dependent not only upon the amount of blood in the cerebral vessels, but also upon its chemical constitution, and especially, it would appear, upon the velocity of the current in the cerebral capillaries. The speed of the blood flow in the cerebral capillaries depends, according to this writer, much more on the tension of the walls of the vessels than upon the height of the arterial pressure. In many of the conditions designated as "cerebral hyperæmia" there is really a condition of lowered pressure, for with flaccidity and widening of the cerebral arteries, due say to paralysis of the sympathetic, the arterial pressure remaining constant, there must follow as the result of the diminution of the tension of the vessel walls a decrease in the velocity of the blood-flow. On the other hand, spasm of the cerebral arteries, due say to irritation of the sympathetic, gives rise not to "anæmia" as generally is supposed, but through increase of vascular tension to a higher velocity of flow through the cerebral capillaries. It has been customary to describe cerebral hyperæmia as being either active or passive.

Thus *active hyperæmia* has been supposed to be associated with febrile conditions, with increased action of the heart, chilling of the surface, contraction of the superficial vessels, and with the suppression of certain customary discharges. Among other recognized causes are plethora, functional irritation, such as is associated with excessive brain work, and the action of certain substances, such as alcohol and nitrite of amyl.

Passive hyperæmia was said to result from obstruction in the cerebral sinuses and veins, engorgement in the lesser circulation, as in mitral stenosis, emphysema, from pressure on the superior cava by aneurisms and tumors, and in the venous engorgement which takes place in prolonged

straining efforts. In its most intense form it is seen in the compression of the superior cava by tumors and in death from strangulation.

The anatomical changes in congestion of the brain are by no means striking. Such an active hyperæmia is never visible post mortem. The veins of the cortex are distended, the gray matter has a deeper color, and its vessels are full. The arteries at the base and in the Sylvian fissures contain blood. Nothing, however, can be more uncertain or indefinite than the post-mortem appearances of so-called hyperæmia of the brain. The most intense distention of the vessels is seen in early death during the specific fevers, or in the secondary passive congestion due to obstruction in the superior cava or in the lesser circulation. In a majority of these cases of so-called hyperæmia, while the total mass of blood in the brain may exceed the normal by a considerable amount, yet the velocity of the current is so much less than normal, that as a result the brain really has a smaller supply of blood than is normal—that is, the patient actually suffers from cerebral "anæmia" rather than from "hyperæmia."

Symptoms.—There are no characteristic or constant features of dilatation of the cerebral blood-vessels. It may exist in the most extreme grade without the slightest disturbance of the cerebral functions, as is witnessed frequently in the pressure by tumors on the superior vena cava. How far the headache and delirium of the early stage of the infectious fevers is to be assigned to dilatation of the blood-vessels of the brain it is not easy to determine. The headache, dizziness, and unpleasant sensations in aortic insufficiency and in some instances of hypertrophy of the heart have been attributed to the cerebral congestion.

As a separate clinical entity, congestion of the brain rarely comes under observation. I have no knowledge of instances associated with delirium, fever, insomnia, and convulsions, or of the so-called apoplectiform variety described by some writers. Very plethoric persons are subject to attacks of headache with flushing of the face and irritability of temper, attacks which may recur frequently and are sometimes relieved by bleeding at the nose. These have usually been attributed to congestion of the brain. When the so-called passive hyperæmia reaches a high grade, there may be torpor, dulness of the intellect, and ultimately deep coma.

Leube suggests that the symptoms usually referred to active hyperæmia in the acute infectious diseases, like diphtheria and erysipelas, or in the instances in which hypertrophy of the heart accompanies disease of the kidneys, may after all be toxic in origin, rather than due to alteration in the circulatory relations. At any rate, he believes that it is not possible to make a diagnosis of such a hyperæmia. Flushing of the face is by no means a safe guide. Possibly an examination of the eye-grounds may be helpful.

2. Anæmia.

This may be induced by loss of blood, either quickly, as in hæmorrhage, or gradually, as in the severe primary and secondary anæmias. The anæmia may be local and due to causes which interfere with the blood supply to the brain, as narrowing of the vessels by endarteritis, pressure,

narrowing of the aortic orifice, or it may follow an unequal distribution of the blood in consequence of dilatation of certain vascular territories. Thus, rapid distention of the intestinal vessels, such as occurs after the removal of ascitic fluid, may cause sudden death from cerebral anæmia. The commonest illustration of this is the fainting fit from emotion, in which the blood supply to the brain is insufficient on account of the diminished arterial pressure. Anæmia of the cerebral vessels may be caused by pressure of fluid in the ventricles. The partial anæmia results from obliteration of branches of the circle of Willis by embolism or thrombosis. Ligature of one carotid sometimes causes a transient marked anæmia and disturbance of function on one side of the brain.

The anatomical condition of the brain in anæmia is very striking. The membranes are pale, only the large veins are full, the small vessels over the gyri are empty, and an unusual amount of cerebro-spinal fluid is present. On section both the gray and white matter look extremely pale and the cut surface is moist. Very few *puncta vasculosa* are seen.

Symptoms.—The effects of anæmia of the brain are well illustrated by a fainting fit in which loss of consciousness follows the heart weakness. When the result of hæmorrhage, there are drowsiness, giddiness, inability to stand, flashes of light, dark spots before the eyes, and noises in the ears; the respiration becomes hurried; the skin is cool and covered with sweat; the pupils are dilated, there may be vomiting, headache, or delirium, and gradually, if the bleeding continues, consciousness is lost and death may occur with convulsions. In ordinary syncope the loss of consciousness is usually transient and the recumbent posture alone may suffice to restore the patient to consciousness. In the more chronic forms of brain anæmia, such as result from the gradual impoverishment of the blood, as in protracted illness or in starvation, the condition known as irritable weakness results. Mental effort is difficult, the slightest irritation is followed by undue excitement, the patient complains of giddiness and noises in the ears, or there may be hallucinations or delirium. These symptoms are met with in an extreme grade as a result of prolonged starvation.

These symptoms are indistinguishable from those due to the so-called cerebral hyperæmia. The quality of the blood is deteriorated and the velocity of the blood-flow is diminished, so that the cerebral nutrition is interfered with. It is interesting to note that lack of suitable nutrition gives rise to phenomena of increased irritability in certain of the cerebral centres, at least for a time.

An interesting set of symptoms, to which the term *hydrencephaloid* was applied by Marshall Hall, occurs in the debility produced by prolonged diarrhœa in children. The child is in a semi-comatose condition with the eyes open, the pupils contracted, and the fontanelle depressed. In the earlier period there may be convulsions. The coma may gradually deepen, the pupils become dilated, and there may be strabismus and even retraction of the head, symptoms which closely simulate those of basilar meningitis.

3. Œdema of the Brain.

In the pathology of brain lesions œdema formerly played a *rôle* almost equal in importance to congestion. It occurs under the following conditions: In general atrophy of the convolutions, in which case the œdema is represented by an increase in the cerebro-spinal fluid and in that of the meshes of the pia. In extreme venous dilatation from obstruction, as in mitral stenosis or in tumors, there may be a condition of congestive œdema, in which, in addition to great filling of the blood-vessels, the substance of the brain itself is unusually moist. The most acute œdema is a local process found around tumors and abscesses. An intense infiltration, local or general, may occur in Bright's disease, and to it, as Traube suggested, certain of the uræmic symptoms may be due.

The *anatomical changes* are not unlike those of anæmia. When the œdema follows progressive atrophy, the fluid is chiefly within and beneath the membranes. The brain substance is anæmic and moist, and has a wet, glistening appearance, which is very characteristic. In some instances the œdema is more intense and local and the brain substance may look infiltrated with fluid. The amount of fluid in the ventricles is usually increased.

The *symptoms* are in great part those of lessened blood-flow, and are not well defined. As just stated, some of the cerebral features of uræmia may depend upon it. Of late years cases have been reported by Raymond, Tenneson, and Dercum, in which unilateral convulsions or paralysis have occurred in connection with chronic Bright's disease, and in which the condition appeared to be associated with œdema of the brain. The older writers laid great stress upon an apoplexia serosa, which may really have been a general œdema of the brain. Inasmuch as the instances in which œdema of the brain occurs are often those in which there is also intoxication, or anæmia, or both, it is probably impossible to say at the bedside definitely which of these possible factors is responsible for the symptoms in a given case.

4. Cerebral Hæmorrhage.

The bleeding may come from branches of either of the two great groups of cerebral vessels—the *basal*, comprising the circle of Willis and the central arteries passing from it and from the first portion of the cerebral arteries, or the *cortical group*, the anterior, middle, and the posterior cerebral vessels. In a majority of the cases the hæmorrhage is from the central branches, more particularly from those given off by the middle cerebral arteries in the anterior perforated spaces, and which supply the corpora striata and internal capsules. One of the largest of these branches which passes to the third division of the lenticular nucleus and to the anterior part of the internal capsule, the lenticulo-striate artery of Duret, is so frequently involved in hæmorrhage that it has been called by Charcot *the artery of cerebral hæmorrhage*. Hæmorrhages from this and from the lenticulo-thalamic artery include more than 60 per cent of all cerebral hæmorrhages. The bleeding may be into the substance of the brain, to which

alone the term cerebral apoplexy is applied, or into the membranes, in which case it is termed meningeal hæmorrhage; both, however, are usually included under the terms intracranial or cerebral hæmorrhage.

Etiology.—The conditions which produce lesions of the blood-vessels play a very important part; thus the natural tendency to degeneration of the vessels in advanced life makes apoplexy much more common after the fiftieth year. It may, however, occur in children under ten. On account of the greater liability to arterial disease (associated probably with muscular exertion and the abuse of alcohol), men are more subject to cerebral hæmorrhage than women. Heredity was formerly thought to be an important factor in this affection, and the apoplectic *habitus* or build is still referred to. By this is meant a stout plethoric body of medium size, with a short neck. Heredity influences cerebral hæmorrhage entirely through the arteries, and there are families in which these degenerate early, usually in association with renal changes. The secondary hypertrophy of the heart brings with it serious dangers, which have already been discussed in the section upon arteries. The special factors in inducing arteriosclerosis—the abuse of alcohol, immoderate eating, syphilis, and prolonged muscular exertion—are found to be important antecedents in a large number of cases of cerebral hæmorrhage. Chronic lead poisoning and gout also may here be mentioned.

The endocarditis of rheumatism and other fevers may indirectly lead to apoplexy by causing embolism and aneurism of the vessels of the brain. Cerebral hæmorrhage occurs occasionally in the specific fevers and in profound alterations of the blood, as in leukæmia and pernicious anæmia. The actual exciting cause of the hæmorrhage is not evident in the majority of cases. The attack may be sudden and without any preliminary symptoms. In other instances violent exertion, particularly straining efforts, or the excited action of the heart in emotion may cause a rupture.

Morbid Anatomy.—The lesions causing apoplexy are almost invariably in the cerebral arteries, in which the following changes may lead directly to it:

(*a*) The production of miliary aneurisms, rupture of which is the most common cause of cerebral hæmorrhage. The origin of the miliary aneurisms is disputed. Charcot thought they resulted from changes in the *adventitia* (periarteritis). Others, with Eichler, Ziegler, and Birch-Hirschfeld, find the primary change in the *intima*. The weight of opinion at present, however, is on the side of the view that the *media* is first degenerated (Roth, Loewenthal). They occur most frequently on the central arteries, but also on the smaller branches of the cortical vessels. On section of the brain substance they may be seen as localized, small dark bodies, about the size of a pin's head. Sometimes they are seen in numbers upon the arteries when carefully withdrawn from the anterior perforated spaces. According to Charcot and Bouchard, who have described them, they are most frequent in the central ganglia. In apoplexy after the fortieth year if sought for they are rarely missed. The actual miliary aneurism, which by its rupture has occasioned the hæmorrhage, may be difficult to find, but if one pours water carefully on the area of hæmorrhage, or, better

still, submerges the apoplectic mass for a time, it will usually be found possible to do so, and even to find the hole in its wall.

(*b*) Aneurism of the branches of the circle of Willis. These are by no means uncommon, and will be considered subsequently.

(*c*) Endarteritis and periarteritis in the cerebral vessels most commonly lead to apoplexy by the production of aneurisms, either miliary or coarse. There are instances in which the most careful search fails to reveal anything but diffuse degeneration of the cerebral vessels, particularly of the smaller branches; so that we must conclude that spontaneous rupture may occur without the previous formation of aneurism.

(*d*) Increased permeability of the walls of the vessels may account for hæmorrhages by *diapedesis* without actual rupture. Such hæmorrhages are not uncommon in cases of contracted kidney, grave anæmia, and various infections and intoxications.

The hæmorrhage may be meningeal, cerebral, or intraventricular.

Meningeal hæmorrhage may be outside the dura, between this membrane and the bone, or between the dura and arachnoid, or between the arachnoid and the pia mater. The following are the chief causes of this form of hæmorrhage: Fracture of the skull, in which case the blood usually comes from the lacerated meningeal vessels, sometimes from the torn sinuses. In these cases the blood is usually outside the dura or between it and the arachnoid. The next most frequent cause is rupture of aneurisms on the larger cerebral vessels. The blood is usually subarachnoid. An intracerebral hæmorrhage may burst into the meninges. A special form of meningeal hæmorrhage is found in the new-born, associated with injury during birth. And lastly, meningeal hæmorrhage may occur in the constitutional diseases and fevers. The blood may be in a large quantity at the base; in cases of ruptured aneurism, particularly, it may extend into the cord or upon the cortex. Owing to the greater frequency of the aneurisms in the middle cerebral vessels, the Sylvian fissures are often distended with blood.

Intracerebral hæmorrhage is most frequent in the neighborhood of the corpus striatum, particularly toward the outer section of the lenticular nucleus. The hæmorrhage may be small and limited to the lenticular body, the thalamus, and the internal capsule, or it may extend into the centrum semi-ovale, or burst into the lateral ventricle, or extend to the insula. Hæmorrhages confined to the white matter—the centrum semi-ovale—are rare. Localized bleeding may occur in the crura or in the pons. Hæmorrhage into the cerebellum is not uncommon, and usually comes from the superior cerebellar artery. The extravasation may be limited to the substance or rupture into the fourth ventricle. Twice I have known sudden death in girls under twenty-five to be due to cerebellar hæmorrhage.

Ventricular Hæmorrhage.—This occasionally but rarely is primary, coming from the vessels of the plexuses or of the walls. More often it is secondary, following hæmorrhage into the cerebral substance. It is not infrequent in early life and may occur during birth. Of 94 cases collected by Edward Sanders, 7 occurred during the first year, and 14 under the twentieth year. In the cases which I have seen in adults it has almost

always been caused by rupture of a vessel in the neighborhood of the caudate nucleus. The blood may be found in one ventricle only, but more commonly it is in both lateral ventricles, and may pass into the third ventricle and through the aqueduct of Sylvius into the fourth ventricle, forming a complete mould in blood of the ventricular system. In these cases the clinical picture may be that of "*apoplexie foudroyante.*"

Subsequent Changes.—The blood gradually changes in color, and ultimately the hæmoglobin is converted into the reddish-brown hæmatoidin. Inflammation occurs about the apoplectic area, limiting and confining it, and ultimately a definite wall may be produced, inclosing a cyst with fluid contents. In other instances a cyst is not formed, but the connective tissue proliferates and leaves a pigmented scar. In meningeal hæmorrhage the effused blood may be gradually absorbed and leave only a staining of the membranes. In other cases, particularly in infants, when the effusion is cortical and abundant, there may be localized wasting of the convolutions and the production of a cyst in the meninges. Possibly certain of the cases of porencephaly are caused in this way.

Secondary degeneration follows, varying in character according to the location of the hæmorrhage and the actual damage done by it to nerve cells or their medullated axones. Thus, in persons dying some years after a cerebral apoplexy which has produced hemiplegia (lesion of the motor area in the cortex or of the pyramidal tract leading from it), the degeneration may be traced through the cerebral peduncle, the ventral part of the pons, the pyramids of the medulla, the fibres of the direct pyramidal tract of the cord of the same side, and the fibres of the crossed pyramidal tract on the opposite side. After hæmorrhages in the middle and inferior frontal gyri there follows degeneration of the frontal cerebro-cortico-pontal path, going through the anterior limb of the internal capsule and the medial portion of the basis pedunculi to the nuclei pontis; also degeneration of the fibres connecting the nucleus medialis thalami, and the anterior part of the nucleus lateralis thalami with the cortex (Flechsig, v. Monakow).

When the temporal gyri or their white matter are destroyed by a hæmorrhage the lateral segment of the basis pedunculi degenerates (Dejerine). Cerebellar hæmorrhage, especially if it injure the nucleus dentatus, may lead to degeneration of the brachium conjunctivum.

There may be slow degeneration in the lemniscus medialis, extending as far as the nuclei on the opposite side of the medulla oblongata, after hæmorrhages in the central gyri, hypothalamic region, or dorsal part of the pons. Hæmorrhages destroying the occipital cortex, or subcortical hæmorrhages injuring the optic radiations, occasion slow degeneration (cellulipetal) of the radiations from the lateral geniculate body, and after a time to marked atrophy or even disappearance of its ganglion cells.

Symptoms.—These may be divided into primary, or those connected with the onset, and secondary, or those which develop later after the early manifestations have passed away.

Primary Symptoms.—Premonitory indications are rare. As a rule, the patient is seized while in full health or about the performance of some every-day action, occasionally an action requiring strain or extra exertion.

Now and then instances are found in which there are sensations of numbness or tingling or pains in the limbs, or even choreiform movements in the muscles of the opposite side, the so-called prehemiplegic chorea. In other cases temporary disturbances of vision and of associated movements of the eye-muscles have been noted, but none of the prodromata of apoplexy (the so-called "warnings") is characteristic. The onset of the apoplexy, as cerebral hæmorrhage is usually called, varies greatly. There may be sudden loss of consciousness and complete relaxation of the extremities. In such instances the name *apoplectic stroke* is particularly appropriate. In other cases the onset is more gradual and the loss of consciousness may not occur for a few minutes after the patient has fallen, or after the paralysis of the limbs is manifest. In the typical apoplectic attack the condition is as follows: There is deep unconsciousness; the patient cannot be roused. The face is injected, sometimes cyanotic, or of an ashen-gray hue. The pupils vary; usually they are dilated, sometimes unequal, and always, in deep coma, inactive. If the hæmorrhage be so located that it can irritate the nucleus of the third nerve the pupils are contracted (hæmorrhages into the pons or ventricles). The respirations are slow, noisy, and accompanied with stertor. Sometimes the Cheyne-Stokes rhythm may be present. The chest movements on the paralyzed side may be restricted, in rare instances on the opposite side. The cheeks are often blown out during expiration, with spluttering of the lips. The pulse is usually full, slow, and of increased tension. The temperature may be normal, but is often found subnormal, and, as in a case reported by Bastian, may sink below 95°. In cases of basal hæmorrhage the temperature, on the other hand, may be high. The urine and fæces are usually passed involuntarily. Convulsions are not common. It may be difficult to decide whether the condition is apoplexy associated with hemiplegia or sudden coma from other causes. An indication of hemiplegia may be discovered in the difference in the tonus of the muscles on the two sides. If the arm or the leg is lifted, it drops "dead" on the affected side, while on the other it falls more slowly. Rigidity also may be present. In watching the movements of the facial muscles in the stertorous respiration it will be seen that on the paralyzed side the relaxation permits the cheek to be blown out in a more marked manner. The head and eyes may be turned strongly to one side—conjugate deviation. In such an event the turning is *toward* the side of the hæmorrhage.

In other cases, in which the onset is not so abrupt, the patient may not lose consciousness, but in the course of a few hours there is loss of power, unconsciousness gradually develops, and deepens into profound coma. This is sometimes termed ingravescent apoplexy. The attack may occur during sleep. The patient may be found unconscious, or wakes to find that the power is lost on one side. Small hæmorrhages in the territory of the central arteries may cause hemiplegia without loss of consciousness.

Usually within forty-eight hours after the onset of an attack, sometimes within from two to six hours, there is febrile reaction, and more or less constitutional disturbance associated with inflammatory changes about the hæmorrhage and absorption of the blood. The period of inflammatory reaction may continue for from one week to two months. The patient may

die in this reaction, or, if consciousness has been regained, there may be delirium or recurrence of the coma. At this period the so-called early rigidity may develop in the paralyzed limbs. The so-called trophic changes may occur, such as sloughing or the formation of vesicles. The most serious of these is the sloughing eschar of the lower part of the back, or on the paralyzed side, which may appear within forty-eight hours of the onset and is usually of grave significance. The congestion at the bases of the lungs so common in apoplexy is regarded by some as a trophic change.

Conjugate Deviation.—In a right hemiplegia the eyes and head may be turned to the left side; that is to say, the eyes look toward the cerebral lesion. This is almost the rule in the conjugate deviation of the head and eyes which occurs early in hemiplegia. When, however, convulsions or spasm develop or the state of so-called early rigidity in hemiplegia, the conjugate deviation of the head and eyes may be in the opposite direction; that is to say, the eyes look away from the lesion and the head is rotated toward the convulsed side. This symptom may be associated with cortical lesions, particularly, according to some authors, when in the neighborhood of the supramarginal and angular gyri. It may also occur in a lesion of the internal capsule or in the pons, but in the latter situation the conjugate deviation is the reverse of that which occurs in other cases, as the patient looks away from the lesion, and in spasm or convulsion looks toward the lesion. In cases in which consciousness is restored and the patient improves, the unilateral paralysis which persists in cases in which the motor area, or the pyramidal tract in any part of its course, is involved is known as

Hemiplegia.—Hemiplegia is complete when it involves face, arm, and leg, or partial when it involves only one or other of these parts. This may be the result of a lesion (*a*) of the motor cortex; (*b*) of the pyramidal fibres in the corona radiata and in the internal capsule; (*c*) of a lesion in the cerebral peduncle; or (*d*) in the pons Varolii. The situation of the lesions and their effects are given in Fig. 10. Hæmorrhage is perhaps the most common cause, but tumors and spots of softening may also induce it. The special details of the hemiplegia may here be considered. The face (except in lesions in the lower part of the pons) is involved on the same side as the arm and leg. This results from the fact that the facial muscles stand in precisely the same relation to the cortical centres as those of the arm and leg, the fibres of the upper motor segment of the facial nerve from the cortex decussating just as do those of the nerves of the limbs. The facial paralysis is partial, involving only the lower portion of the nerve, so that the orbicularis oculi and the frontalis muscles are uninvolved. The signs of the facial paralysis are usually well marked. There may be a slight difficulty in elevating the eyebrows or in closing the eye on the paralyzed side, or in rare cases the facial paralysis is complete, but the movements may be present with emotion, as laughing or crying. The hypoglossal nerve also is involved. In consequence, the patient cannot put out the tongue straight, but it deviates toward the paralyzed side, inasmuch as the genio-hyo-glossus of the sound side is unopposed. With right hemiplegia there may be aphasia. Even without marked aphasia difficulty in speaking and slowness are common.

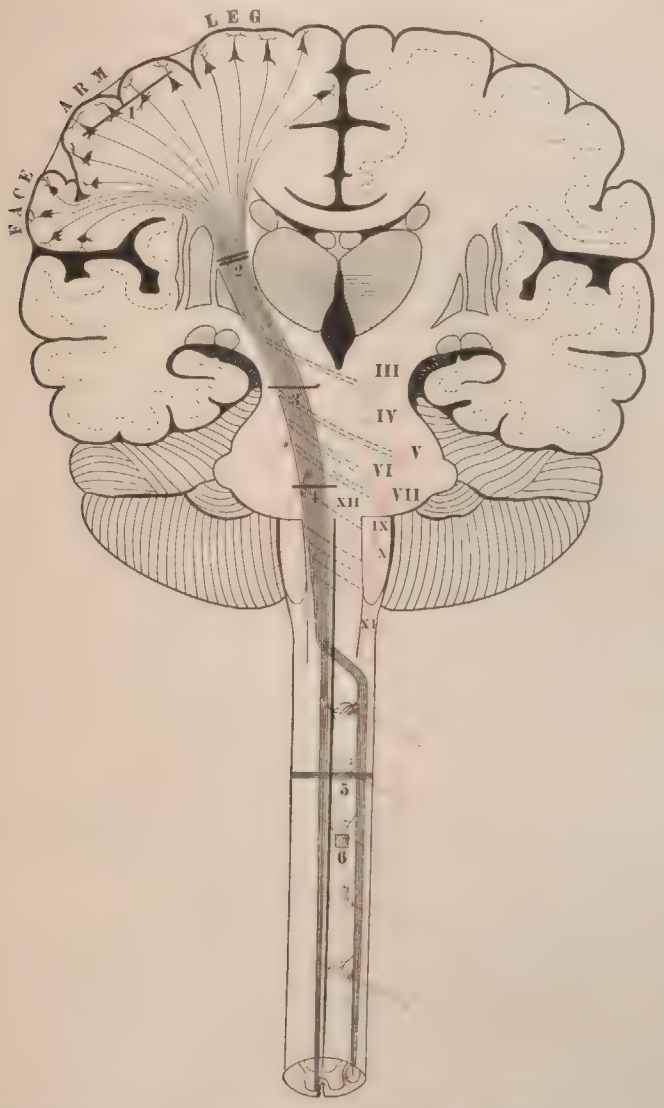

FIG. 10.—Diagram of motor path from right brain. The upper segment is black, the lower red. The nuclei of the motor cerebral nerves are shown on the left side; on the right side the cerebral nerves of that side are indicated. A lesion at 1 would cause upper segment paralysis in the arm of the opposite side—cerebral monoplegia; at 2, upper segment paralysis of the whole opposite side of the body—hemiplegia; at 3 (in the crus), upper segment paralysis of the opposite face, arm and leg, and lower segment paralysis of the eye muscles on the same side—crossed paralysis; at 4 (in the lower part of the pons), upper segment paralysis of the opposite arm and leg, and lower segment paralysis of the face and the external rectus on the same side—crossed paralysis; at 5, upper segment paralysis of all muscles represented below lesion, and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6, lower segment paralysis of muscles localized at seat of lesion—anterior poliomyelitis. (Van Gehuchten, modified.)

The arm is, as a rule, more completely paralyzed than the leg. The loss of power may be absolute or partial. In severe cases it is at first complete. In others, when the paralysis in the face and arm is complete that of the leg is only partial. The face and arm may alone be paralyzed, while the leg escapes. Less commonly the leg is more affected than the arm, and the face may be only slightly involved.

Certain muscles escape in hemiplegia, particularly those associated in symmetrical movements, as those of the thorax and abdomen, a fact which Broadbent explains by supposing that as the spinal nuclei controlling these movements on both sides constantly act together, they may, by means of this intimate connection, be stimulated by impulses coming from only one side of the brain. The degree of permanent paralysis after a hemiplegic attack varies much in different cases. When the restitution is partial, it is always, as Wernicke has pointed out, certain groups of muscles which recover rather than others. Thus in the leg the residual paralysis concerns the flexors of the leg and the dorsal flexors of the foot—i. e., the muscles which, according to Ludwig Mann, are active in the second period of walking, shortening the leg, and bringing it forward while it swings. The muscles which lift the leg when it rests upon the ground, those used in the first period of walking, include the extensors of the leg and the plantar flexors of the foot. These "lengtheners" of the leg often recover almost completely in cases in which the paralysis is due to lesions of the pyramidal tract. In the arms the residual paralysis usually affects the muscle groups which oppose the thumb, those which rotate the arm outward, and the openers of the hand.

As a rule, there is at first no wasting of the paralyzed limbs.

Crossed Hemiplegia.—A paralysis in which there is loss of function in a cerebral nerve on one side with loss of power (or of sensation) on the opposite side of the body is called a crossed or alternate hemiplegia. It is met with in lesions, commonly hæmorrhage, in the crus, the pons, and the medulla (Fig, 10, 3 and 4).

(*a*) *Crus.*—The bleeding may extend from vessels supplying the corpus striatum, internal capsule, and optic thalamus, or the hæmorrhage may be primarily in the crus. In the classical case of Weber, on section of the lower part of the left crus an oblong clot 15 mm. in length lay just below the medial and inferior surface. The characteristic features of a lesion in this locality are paralysis of arm, face, and leg of the opposite side, and oculo-motor paralysis of the same side—the syndrome of Weber. Sensory changes have also been present. Hæmorrhage into the tegmentum is not necessarily associated with hemiplegia, but there may be incomplete paralysis of the oculo-motor nerve, with disturbance of sensation and ataxia on the opposite side of the body. The optic tract or the lateral geniculate body lying on the lateral side of the crus may be compressed, in which event there will be hemianopsia.

(*b*) *Pons and Medulla.*—Lesions may involve the pyramidal tract and one or more of the cerebral nerves. If at the lower aspect of the pons, the facial nerve may be involved, causing paralysis of the face on the same side and hemiplegia on the opposite side. The fifth nerve may be involved,

with the fillet (the sensory tract), causing loss of sensation in the area of distribution of the fifth on the same side as the lesion and loss of sensation on the opposite side of the body.

Sensory Disturbances resulting from Cerebral Hæmorrhage.—These are variable. Hemianæsthesia may coexist with hemiplegia, but in many instances there is only slight numbing of sensation. When the hemianæsthesia is marked, it is usually the result of a lesion in the internal capsule involving the retrolenticular portion of the posterior limb. In C. L. Dana's study of sensory localization he found that anæsthesia of organic cortical origin was always limited or more pronounced in certain parts, as the face, arm, or leg, and was generally incomplete. Total anæsthesia was either of functional or subcortical origin. Marked anæsthesia was much more common in softening than in hæmorrhage. Complete hemianæsthesia is certainly rare in hæmorrhage. Disturbance of the special senses is not common. Hemianopia may exist on the same side as the lesion, and there may be diminution in the acuteness of the senses of hearing, taste, and smell. Gowers thinks that homonymous hemianopsia of the halves of the visual fields opposite to the lesion is very frequent, though often overlooked.

Psychic disturbances, variable in nature and degree, may result from cerebral hæmorrhage.

The Reflexes in Apoplectic Cases.—During the apoplectic coma all the reflexes are abolished, but immediately on recovery of consciousness they return, first on the non-hemiplegic side, later, sometimes only after weeks, on the paralyzed side. As to the time of return, especially of the patellar reflexes, marked differences are observable in individual cases. The deep reflexes later are increased on the paralyzed side, and ankle clonus may be present. The plantar and other superficial reflexes are usually diminished. The sphincters are not affected.

The *course* of the disease depends upon the situation and extent of the lesion. If slight, the hemiplegia may disappear completely within a few days or a few weeks. In severe cases the rule is that the leg gradually recovers before the arm, and the muscles of the shoulder girdle and upper arm before those of the forearm and hand. The face may recover quickly.

Except in the very slight lesions, in which the hemiplegia is transient, changes take place which may be grouped as

Secondary Symptoms.—These correspond to the chronic stage. In a case in which little or no improvement takes place within eight or ten weeks, it will be found that the paralyzed limbs undergo certain changes. The leg, as a rule, recovers enough power to enable the patient to get about, although the foot is dragged. Occasionally a recurrence of severe symptoms is seen, even without a new hæmorrhage having taken place. In both arm and leg the condition of *secondary contraction* or *late rigidity* comes on and is always most marked in the upper extremity. The arm becomes permanently flexed at the elbow and resists all attempts at extension. The wrist is flexed upon the forearm and the fingers upon the hand. The position of the arm and hand is very characteristic. There is frequently, as the contractures develop, a great deal of pain. In the leg the contracture is

rarely so extreme. The loss of power is most marked in the muscles of the foot, and to prevent the toes from dragging, the knee in walking is much flexed, or more commonly the foot is swung round in a half-circle.

The reflexes are at this stage greatly increased. These contractures are permanent and incurable, and are associated with a secondary descending sclerosis of the motor path. There are instances, however, in which rigidity and contracture do not occur, but the arm remains flaccid, the leg having regained its power. This *hémiplégie flasque* of Bouchard is found most commonly in children. Among other secondary changes in late hemiplegia may be mentioned the following: Tremor of the affected limbs, post-paralytic chorea, the mobile spasm known as athetosis, arthropathies in the joints of the affected side, and muscular atrophy. Athetosis and post-hemiplegic chorea will be considered in the hemiplegia of children. The cool surface and thin glossy skin of a hemiplegic limb are familiar to all. A word may here be said upon the subject of muscular atrophy of cerebral origin.

As a rule, atrophy is not a marked feature in hemiplegia, but in some instances it does develop. It has been thought to be due in some cases to secondary alterations in the gray matter of the ventral horns, as in a case reported by Charcot. Recently, however, attention has been called by Senator, Quincke, and others to the fact that atrophy may follow as a direct result of the cerebral lesion, the ventral horns remaining intact. In Quincke's case, atrophy of the arm followed the development of a glioma in the anterior central convolution. The gray matter of the ventral horns was normal. These atrophies are most common in cortical lesions involving the domain of the third main branch of the Sylvian artery, and in central lesions involving the lenticulo-thalamic region. Their explanation is not clear. The wasting of cerebral origin, which occurs most frequently in children, and leads to hemiatrophy of the muscles along with stunted growth of the bones and joints, is to be sharply separated from the hemiatrophy of the muscles of the adult following within a relatively short time upon the hemiplegia.

Diagnosis.—There are three groups of cases which offer increasing difficulty in recognition.

(1) Cases in which the onset is gradual, a day or two elapsing before the paralysis is fully developed and consciousness completely lost, are readily recognized, though it may be difficult to determine whether the lesion is due to thrombosis or to hæmorrhage.

(2) In the sudden apoplectic stroke in which the patient rapidly loses consciousness, the difficulty in diagnosis may be still greater, particularly if the patient is in deep coma when first seen.

The first point to be decided is the existence of hemiplegia. This may be difficult, although, as a rule, even in deep coma the limbs on the paralyzed side are more flaccid and drop instantly when lifted; whereas, on the non-paralyzed side the muscles retain some degree of tonus. The reflexes may be increased on the affected side and there may be conjugate deviation of the head and eyes. Rigidity in the limbs of one side is in favor of a

hemiplegic lesion. It is practically impossible in a majority of these cases to say whether the lesion is due to hæmorrhage, embolism, or thrombosis.

(3) Large hæmorrhage into the ventricles or into the pons may produce sudden loss of consciousness with complete relaxation, so that the condition may simulate coma from uræmia, diabetes, alcoholism, opium poisoning, or epilepsy.

The previous history and the mode of onset may give valuable information. In epilepsy, convulsions have preceded the coma; in alcoholism, there is a history of constant drinking, while in opium poisoning the coma develops more gradually; but in many instances the difficulty is practically very great, and on more than one occasion I have seen mortifying post-mortem disclosures under these circumstances. With diabetic coma the breath often smells of acetone. In ventricular hæmorrhage the coma is sudden and develops rapidly. The hemiplegic symptoms may be transient, quickly giving place to complete relaxation. Convulsions occur in many cases, and may be the very symptom to lead astray—as in a case of ventricular hæmorrhage which occurred in a puerperal patient, in whom, naturally enough, the condition was thought to be uræmic. Rigidity is often present. In hæmorrhage into the pons convulsions are frequent. The pupils may be strongly contracted, conjugate deviation may occur, and the temperature is apt to rise rapidly. The contraction of the pupils in pontine hæmorrhage naturally suggests opium poisoning. The difference in temperature in the two conditions is a valuable diagnostic point. The apoplectiform seizures of general paresis have usually been preceded by abnormal mental symptoms, and the associated hemiplegia is seldom permanent.

It may be impossible at first to give a definite diagnosis. In admissions to hospitals or in emergency cases the physician should be particularly careful about the following points: The examination of the head for injury or fracture; the urine should be tested for albumin, examined for sugar, and studied microscopically; a careful examination should be made of the limbs with reference to their degree of relaxation or the presence of rigidity, and the condition of the reflexes; the state of the pupils should be noted and the temperature taken. The odor of the breath (alcohol, acetone, chloroform, etc.) should be remarked. The most serious mistakes are made in the case of patients who are drunk at the time of the attack, a combination by no means uncommon in the class of patients admitted to hospital. Under these circumstances the case may erroneously be looked upon as one of alcoholic coma. It is best to regard each case as serious and to bear in mind that this is a condition in which, above all others, mistakes are common.

Prognosis.—From cortical hæmorrhage, unless very extensive, the recovery may be complete without a trace of contracture. This is more common when the hæmorrhage follows injury than when it results from disease of the arteries. Infantile meningeal hæmorrhage, on the other hand, is a condition which may produce idiocy or spastic diplegia.

Large hæmorrhages into the corona radiata, and especially those which rupture into the ventricles, rapidly prove fatal.

The hemiplegia which follows lesions of the internal capsule, the result

of rupture of the lenticulo-striate artery, is usually persistent and followed by contracture. When the retro-lenticular fibres of the internal capsule are involved there may be hemianæsthesia, and later, especially if the thalamus be implicated, hemichorea or athetosis. In any case of cerebral apoplexy the following symptoms are of grave omen: persistence or deepening of the coma during the second and third day; rapid rise in temperature within the first forty-eight hours after the initial fall. In the reaction which takes place on the second or third day, the temperature usually rises, and its gradual fall on the third or fourth day with return of consciousness is a favorable indication. The rapid formation of bed-sores, particularly the malignant decubitus of Charcot, is a fatal indication. The occurrence of albumin and sugar, if abundant, in the urine is an unfavorable symptom.

When consciousness returns and the patient is improving, the question is anxiously asked as to the paralysis. The extent of this cannot be determined for some weeks. With slight lesions it may pass off entirely. If persistent at the end of a month some grade of permanent palsy is certain to remain, and gradually the late rigidity supervenes.

5. Embolism and Thrombosis (*Cerebral Softening*).

(*a*) **Embolism.**—The embolus usually enters the carotid, rarely the vertebral artery. In the great majority of cases it comes from the left heart and is either a vegetation of a fresh endocarditis or, more commonly, of a recurring endocarditis, or from the segments involved in an ulcerative process. Less often the embolus is a portion of a clot which has formed in the auricular appendix. Portions of clot from an aneurism, thrombi from atheroma of the aorta, or from the territory of the pulmonary veins, may also cause blocking of the branches of the circle of Willis. In the puerperal condition cerebral embolism is not infrequent. It may occur in women with heart-disease, but in other instances the heart is uninvolved, and the condition has been thought to be associated with the development of heart-clots, owing to increased coagulability of the blood. A majority of cases of embolism occur in heart-disease, 89 per cent (Saveliew). Cases are rare in the acute endocarditis of rheumatism, chorea, and febrile conditions. It is much more common in the secondary recurring endocarditis which attacks old sclerotic valves. The embolus most frequently passes to the left middle cerebral artery, as it enters the left carotid oftener than the right because of the more direct course of the blood in the former. The posterior cerebral and the vertebral are less often affected. A large plug may lodge at the bifurcation of the basilar. Embolism of the cerebral vessels is rare.

Embolism occurs more frequently in women, owing, no doubt, to the greater frequency of mitral stenosis. Contrary to this general statement, Newton Pitt's statistics of 79 cases at Guy's Hospital indicate, however, that males are more frequently affected; for in this series there were 44 males and 35 females. Saveliew gives 54 per cent in women.

(*b*) **Thrombosis.**—Clotting of blood in the cerebral vessels occurs (1) about an embolus, (2) as the result of a lesion of the arterial wall (either

endarteritis with or without atheroma or, particularly, the syphilitic arteritis), (3) in aneurisms both coarse and miliary, and (4) very rarely as a direct result of abnormal conditions of the blood. Thrombosis occasionally follows ligation of the carotid artery. The thrombosis is most common in the middle cerebral and in the basilar arteries. According to Kolisko, softening of limited areas, sufficient to induce hemiplegia, may be caused by sudden collapse of certain cerebral arteries from cardiac weakness.

Anatomical Changes.—Degeneration and softening of the territory supplied by the vessels is the ultimate result in both embolism and thrombosis. Blocking in a terminal artery may be followed by infarction, in which the territory may either be deeply infiltrated with blood (hæmorrhagic infarction) or be simply pale, swollen, and necrotic (anæmic infarction). Gradually the process of softening proceeds, the tissue is infiltrated with serum and is moist, the nerve fibres degenerate and become fatty. The neuroglia is swollen and œdematous. The color of the softened area depends upon the amount of blood. The hæmoglobin undergoes gradual transformation, and the early red color may give place to yellow. Formerly much stress was laid upon the difference between *red*, *yellow*, and *white* softening. The red and yellow are seen chiefly on the cortex. Sometimes the red softening is particularly marked in cases of embolism and in the neighborhood of tumors. The gray matter shows many punctiform hæmorrhages—capillary apoplexy. There is a variety of yellow softening—the *plaques jaunes*—common in elderly persons, which occurs in the gray matter of the convolutions. The spots are from 1 to 2 cm. in diameter, sometimes are angular in shape, the edges cleanly cut, and the softened area is represented by either a turbid, yellow material, or in some instances there is a space crossed by fine trabeculæ, in the meshes of which there is fluid. White softening occurs most frequently in the white matter, and is seen best about tumors and abscesses. Inflammatory changes are common in and about the softened areas. When the embolus is derived from an infected focus, as in ulcerative endocarditis, suppuration may follow. The final changes vary very much. The degenerated and dead tissue elements are gradually but slowly removed, and if the region is small may be replaced by a growth of connective tissue and the formation of a scar. If large, the resorption results in the formation of a cyst. It is surprising for how long an area of softening may persist without much change.

The position and extent of the softening depend upon the obstructed artery. An embolus which blocks the middle cerebral at its origin involves not only the arteries to the anterior perforated space, but also the cortical branches, and in such a case there is softening in the neighborhood of the corpus striatum, as well as in part of the region supplied by the cortical vessels. The freedom of anastomosis between these branches varies a good deal. Thus, there are instances of embolism of the middle cerebral artery in which the softening has only involved the territory of the central branches, in which case blood has reached the cortex through the anterior and posterior cerebrals. When the middle cerebral is blocked (as is perhaps oftenest the case) beyond the point of origin of the central arteries, one or other of its branches is usually most involved. The embolus may lodge

in the vessel passing to the third frontal convolution, or in the artery of the ascending frontal or ascending parietal; or it may lodge in the branch passing to the supramarginal and angular gyri, or it may enter the lowest branch which is distributed to the upper convolutions of the temporal lobe. These are practically terminal arteries, and instances frequently occur of softening limited to a part, at any rate, of the territory supplied by them. Some of the most accurate focalizing lesions are produced in this way.

Symptoms.—Extensive thrombotic softening may exist without any symptoms. It is not uncommon in the post-mortem examination of the bodies of elderly persons to find the *plaques jaunes* scattered over the convolutions. So, too, softening may take place in the "silent" regions, as they are termed, without exciting any symptoms. When the central or cortical branches of the middle cerebral arteries are involved the symptoms are similar to those of hæmorrhage from the same arteries. Permanent or transient hemiplegia results. When the central arteries are involved the softening in the internal capsule is commonly followed by permanent hemiplegia. There are certain peculiarities associated with embolism and with thrombosis respectively.

In *embolism* the patient is usually the subject of heart-trouble, or there exist some of the conditions already mentioned. The onset is sudden, without premonitory symptoms. When the embolism blocks the left middle cerebral artery the hemiplegia is usually associated with aphasia. In *thrombosis*, on the other hand, the onset is more gradual; the patient has previously complained of headache, vertigo, tingling in the fingers; the speech may have been embarrassed for some days; the patient has had loss of memory or is incoherent, or paralysis begins at one part, as the hand, and extends slowly, and the hemiplegia may be incomplete or variable. Abrupt loss of consciousness is much less common, and when the lesion is small consciousness is retained. Thus, in thrombosis due to syphilitic disease, the hemiplegia may come on gradually without the slightest disturbance of consciousness.

The hemiplegia following thrombosis or embolism has practically the characteristics, both primary and secondary, described under hæmorrhage.

The following may be the effects of blocking the different vessels: (*a*) *Vertebral.*—The left branch is more frequently plugged. The effects are involvement of the nuclei in the medulla and symptoms of acute bulbar paralysis. It rarely occurs alone; more commonly with

(*b*) Blocking of the *basilar artery.* When this is entirely occluded, there may be bilateral paralysis from involvement of both motor paths. Bulbar symptoms may be present; rigidity or spasm may occur. The temperature may rise rapidly. The symptoms, in fact, are those of apoplexy of the pons.

(*c*) The *posterior cerebral* supplies the occipital lobe on its medial surface and the greater part of the temporo-sphenoidal lobe. If the main stem be thrombosed there is hemianopia with sensory aphasia. Localized areas of softening may exist without symptoms. Blocking of the main occipital branch (arteria occipitalis of Duret), or of the arteria calcarina, passing to the cuneus may be followed by hemianopia. Hemianæsthesia may result from involvement of the posterior part of the internal capsule. Not

infrequently symmetrical thrombosis of the occipital arteries of the two sides occurs, as in Förster's well-known case. Still more frequent is the occurrence of thrombosis of a branch of the posterior cerebral of one hemisphere and a branch of the middle cerebral of the other (von Monakow). It is in such cases that the most pronounced instances of apraxia are met with.

(*d*) *Internal Carotid.*—The symptoms are variable. As is well known, the vessel is in a majority of cases ligated without risk. In other instances transient hemiplegia follows; in others again the hemiplegia is permanent. These variations depend on the anastomoses in the circle of Willis. If these are large and free, no paralysis follows, but in cases in which the posterior communicating and the anterior communicating vessels are small or absent, the paralysis may persist. In No. 7 of my Elwyn series of cases of infantile hemiplegia, the woman, aged twenty-four, when six years old, had the right carotid ligated for abscess following scarlet fever, with the result of permanent hemiplegia. Blocking of the internal carotid within the skull by thrombosis or embolism is followed by hemiplegia, coma, and usually death. The clot is rarely confined to the carotid itself, but spreads into its branches and may involve the ophthalmic artery.

(*e*) *Middle Cerebral.*—This is the vessel most commonly involved, and, as already mentioned, if plugged before the central arteries are given off, permanent hemiplegia usually follows from softening of the internal capsule. Blocking of the branches beyond this point may be followed by hemiplegia, which is more likely to be transient, involves chiefly the arm and face, and if on the left side is associated with aphasia. The individual branches passing to the inferior frontal (producing typical motor aphasia if the disease be on the left side), anterior and posterior central gyri (usually causing total hemiplegia), to the supramarginal and angular gyri (giving rise, if the thrombosis be on the left side, probably without exception to the so-called pure (or subcortical) alexia, usually also to right-sided hemianopsia), or to the temporal gyri (in which event with left-sided thrombosis word-deafness results) may be plugged.

(*f*) *Anterior Cerebral.*—No symptoms may follow, and even when the branches which supply the paracental lobule and the top of the ascending convolutions are plugged the branches from the middle cerebral are usually able to effect a collateral circulation in these parts. Monoplegia of the leg may, however, result. Hebetude and dulness of intellect may occur with obstruction of the vessel.

There is unquestionably greater freedom of communication in the cortical branches of the different arteries than is usually admitted, although it is not possible, for example, to inject the posterior cerebral through the middle cerebral, or the middle cerebral from the anterior; but the absence of softening in some instances in which smaller branches are blocked shows how complete may be the compensation, probably by way of the capillaries. The dilatation of the collateral branches may take place very rapidly; thus a patient with chronic nephritis died about twenty-four hours after the hemiplegic attack. There were recent vegetations on the mitral valve and an embolus in the right middle cerebral artery just beyond the first two

branches. The central portion of the hemisphere was swollen and œdematous. The right anterior cerebral was greatly dilated, and by measurement its diameter was found to be nearly three times that of the left.

Treatment of Cerebral Hæmorrhage and of Softening.—The patient should be placed on his back, with the head high, the neck free, kept absolutely quiet, and measures immediately taken to reduce the arterial pressure. Of these the most rapid and satisfactory is venesection, which should be practised whenever the arterial tension is much increased. With a small pulse of low tension and signs of cardiac weakness it is contra-indicated. The chief difficulty is in determining whether the apoplexy is really due to hæmorrhage, or to thrombosis or embolism, since in the latter group of cases bleeding probably does harm. As a rule, however, in middle-aged men with arterio-sclerosis, an accentuated aortic second sound, and hypertrophy of the left ventricle, bleeding is indicated. Horsley and Spencer have recently, on experimental grounds, recommended the practice, formerly employed empirically, of compression of the carotid, particularly in the ingravescent form; or even, in suitable cases, passing a ligature round the vessel. An ice-bag may be placed on the head and hot bottles to the feet. The bowels should be freely opened, either by calomel, or croton oil placed on the tongue. Counter-irritation to the neck or to the feet is not necessary. Catheterization of the bladder may be necessary, especially if the patient remain long unconscious. When dyspnœa, stertor, and signs of mechanical obstruction are present, the patient should be turned on the side, as recommended by Bowles. This procedure also lessens the liability to congestion of the lungs.

Special care should be taken to avoid bed-sores; and if bottles are used to the feet, they should not be too hot, since blisters may be readily caused by much lower temperature than in health. In the fever of reaction, aconite may be indicated, but should be cautiously used. Stimulants are not necessary, unless the pulse becomes feeble and signs of collapse supervene. No digitalis is to be given. During recovery the patient should be still kept entirely at rest, even in the mildest cases remaining in bed for at least fourteen days. The ice-bag should still be kept at the head. The diet should be light and no medicine other than some placebo should be administered, at least during the first month after the hæmorrhage. Attention should be paid to the position occupied by the paralyzed limb or limbs, which if swollen may be wrapped in cotton batting or flannel.

The treatment of *softening* from thrombosis or embolism is very unsatisfactory. Venesection is not indicated, as it lowers the tension and rather promotes clotting. If, as is often the case, the heart's action is feeble and irregular, stimulants and small doses of digitalis may be given with, if necessary, ether or ammonia. The bowels should be kept open, but it is not well to purge actively, as in hæmorrhage.

In the thrombosis which follows syphilitic disease of the arteries, and which is met with most frequently in men between twenty and forty (in whom the hemiplegia often sets in without loss of consciousness), the iodide of potassium should be freely used, giving from 20 to 30 grains three times a day, or, if necessary, larger doses. If the syphilis has been recent, mer-

curials by inunction are also indicated. Practically these are the only cases of hemiplegia in which we see satisfactory results from treatment.

Operative treatment has been suggested, and when the diagnosis of subdural hæmorrhage can be made it is justifiable. An attempt to reach a central hæmorrhage in the neighborhood of the internal capsule would only increase the damage to the brain substance. Very little can be done for the hemiplegia which remains. The damage is too often irreparable and permanent, and it is very improbable that iodide of potassium, or any other remedy, hastens in the slightest degree Nature's dealing with the blood-clot.

The paralyzed limbs may be gently rubbed once or twice a day, and this should be systematically carried out, in order to maintain the nutrition of the muscles and to prevent, if possible, contractures. The massage should not, however, be begun until at least ten days after the attack. The rubbing should be *toward* the body, and should not be continued for more than fifteen minutes at a time. After the lapse of a fortnight, or in severe cases a month, the muscles may be stimulated by the faradic current; faradic stimulation alternating with massage, especially if applied to the antagonists of the muscles which ordinarily undergo contracture, is of very great service, even in cases where there can be but little hope of any return of voluntary movement. When contractures develop, electricity properly applied at intervals may still be of some benefit along with the passive movements and frictions.

In a case of complete hemiplegia, the friends should at the outset be frankly told that the chances of full recovery are slight. Power is usually restored in the leg sufficient to enable the patient to get about, but in the majority of instances the finer movements of the hand are permanently lost. The general health should be looked after, the bowels regulated, and the secretions of the skin and kidneys kept active. In permanent hemiplegia in persons above the middle period of life, more or less mental weakness is apt to follow the attack, and the patient may become irritable and emotional.

And, lastly, when hemiplegia has persisted for more than three months and contractures have developed, it is the duty of the physician to explain to the patient, or to his friends, that the condition is past relief, that medicines and electricity will do no good, and that there is no possible hope of cure.

6. Aneurism of the Cerebral Arteries.

Miliary aneurisms are not included, but reference is made only to aneurism of the larger branches. The condition is not uncommon. There were 12 instances in my first 800 autopsies in Montreal.* This is a considerably larger proportion than in Newton Pitt's collection from Guy's Hospital, 19 times in 9,000 inspections.

Etiology.—Males are more frequently affected than females. Of my 12 cases 7 were males. The disease is most common at the middle period

* Canada Medical and Surgical Journal, vol. xiv.

of life. One of my cases was a lad of six. Pitt describes one at the same age. The chief causes are (*a*) endarteritis, either simple or syphilitic, which leads to weakness of the wall and dilatation; and (*b*) embolism. As pointed out by Church, these aneurisms are often found with endocarditis. Pitt, in his recent study of the subject, concludes that it is exceptional to find cerebral aneurism unassociated with fungating endocarditis. The embolus disappears, and dilatation follows the secondary inflammatory changes in the coats of the vessel.

Morbid Anatomy.—The middle cerebral branches are most frequently involved. In my 12 cases the distribution on the arteries was as follows: Internal carotid, 1; middle cerebral, 5; basilar, 3; anterior communicating, 3. Except in one case they were saccular and communicated with the lumen of the vessel by an orifice smaller than the circumference of the sac. In the 154 cases which make up the statistics of Lebert, Durand, and Bartholow the middle cerebral was involved in 44, the basilar in 41, internal carotid in 23, anterior cerebral in 14, posterior communicating in 8, anterior communicating in 8, vertebral in 7, posterior cerebral in 6, inferior cerebellar in 3 (Gowers). The size of the aneurism varies from that of a pea to that of a walnut. The hæmorrhage may be entirely meningeal with very slight laceration of the brain substance, but the bleeding may be, as Coats has shown, entirely within the substance.

Symptoms.—The aneurism may attain considerable size and cause no symptoms. In a majority of the cases the first intimation is the rupture and the fatal apoplexy. Distinct symptoms are most frequently caused by aneurism of the internal carotid, which may compress the optic nerve or the commissure, causing neuritis or paralysis of the third nerve. A murmur may be audible on auscultation of the skull. Aneurism in this situation may give rise to irritative and pressure symptoms at the base of the brain or to hemianopsia. In the remarkable case reported by Weir Mitchell and Dercum an aneurism compressed the chiasma and produced bilateral temporal hemianopsia.

Aneurism of the vertebral or of the basilar may involve the nerves from the fifth to the twelfth. A large sac at the termination of the basilar may compress the third nerves or the crura.

The diagnosis is, as a rule, impossible. The larger sacs produce the symptoms of tumor, and their rupture is usually fatal.

7. Endarteritis.

In no group of vessels do we more frequently see chronic degenerative changes than in those of the circle of Willis. The condition occurs as:

(*a*) *Arterio-sclerosis*, producing localized or diffused thickening of the intima with the formation of atheromatous patches or areas of calcification. In the later stages, as seen in elderly people, the arteries of the circle of Willis may be dilated, stiff, or almost universally calcified.

(*b*) *Syphilitic Endarteritis.*—As already mentioned under the section of syphilis, gummatous endarteritis is specially prone to attack the cerebral vessels. It has in itself no specific characters—that is to say, it is im-

possible in given sections to pick out an endarteritis syphilitica from an ordinary endarteritis obliterans. On the other hand, as already stated, the nodular periarteritis is never seen except in syphilis.

8. Thrombosis of the Cerebral Sinuses and Veins.

The condition may be primary or secondary. Lebert (1854) and Tonnele were among the first to recognize the condition clinically.

Primary thrombosis of the sinuses and veins is rare. It occurs (*a*) in children, particularly during the first six months of life, usually in connection with diarrhœa. It has, in my experience, been a rare condition. I have never seen an example of spontaneous thrombosis of the sinuses in a child, and only two instances, both in connection with meningitis, in which the cortical veins contained clots. Gowers believes that it is of frequent occurrence, and that thrombosis of the veins is not an uncommon cause of infantile hemiplegia.

(*b*) In connection with chlorosis and anæmia, the so-called *autochthonous sinus-thrombosis*. Brayton Ball has called attention to this interesting association, and has reported 1 case and collected 10 or 11 others from the literature. All were in girls with anæmia or chlorosis. The longitudinal sinus is most frequently involved. The thrombosis of the cerebral sinuses in such cases is usually associated with venous thromboses in other parts of the body, and the patients die, as a rule, in from one to three weeks.

(*c*) In the terminal stages of cancer, phthisis, and other chronic diseases thrombosis may gradually occur in the sinuses and cortical veins. To the coagulum developing in these conditions the term marantic thrombus is applied.

Secondary thrombosis is much more frequent and follows extension of inflammation from contiguous parts to the sinus wall. The common causes are disease of the internal ear, fracture, compression of the sinuses by tumor, or suppurative disease outside the skull, particularly erysipelas, carbuncle, and parotitis. In secondary cases the lateral sinus is most frequently involved. Of 57 fatal cases in which ear-disease caused death with cerebral lesions, there were 22 in which thrombosis existed in the lateral sinuses (Pitt). Tuberculous caries of the temporal bone is often directly responsible. The thrombus may be small, or may fill the entire sinus and extend into the internal jugular vein. In more than one half of these instances the thrombus was suppurating. The disease spreads directly from the necrosis on the posterior wall of the tympanum. According to Voltolini, the inflammation extends by way of the petroso-mastoid canal. It is not so common in disease of the mastoid cells.

Symptoms.—Primary thrombosis of the longitudinal sinus may occur without exciting symptoms and is found accidentally at the post mortem. There may be mental dulness with headache. Convulsions and vomiting may occur. In other instances there is nothing distinctive. In a patient who died under my care, at the Philadelphia Hospital, of phthisis, there was a gradual torpor, deepening to coma, without convulsions, localizing symptoms, or optic neuritis. The condition was thought to be due to a

terminal meningitis. In the chlorosis cases the head symptoms have, as a rule, been marked. Ball's patient was dull and stupid, had vomiting, dilatation of the pupils, and double choked disks. Slight paresis of the left side occurred. An interesting feature in her case was the development of swelling of the left leg. In the cases reported by Andrews, Church, Tuckwell, Isambard Owen, and Wilks the patients had headache, vomiting, and delirium. Paralysis was not present. In Douglas Powell's case, with similar symptoms, there was loss of power on the left side. Bristowe reports a case of great interest in an anæmic girl of nineteen, who had convulsions, drowsiness, and vomiting. Tenderness and swelling developed in the position of the right internal jugular vein, and a few days later on the opposite side. The diagnosis was rendered definite by the occurrence of phlebitis in the veins of the right leg. The patient recovered.

The onset of such symptoms as have been mentioned in an anæmic or chlorotic girl should lead to the suspicion of cerebral thrombosis. In infants the diagnosis can rarely be made. Involvement of the cavernous sinus may cause œdema about the eyelids or prominence of the eyes.

In the *secondary thrombi* the symptoms are commonly those of septicæmia. For instance, in over 70 per cent of Pitt's cases the mode of death was by pulmonary pyæmia. This author draws the following important conclusions: (1) The disease spreads oftener from the posterior wall of the middle ear than from the mastoid cells. (2) The otorrhœa is generally of some standing, but not always. (3) The onset is sudden, the chief symptoms being pyrexia, rigors, pains in the occipital region and in the neck, associated with a septicæmic condition. (4) Well-marked optic neuritis may be present. (5) The appearance of acute local pulmonary mischief or of distant suppuration is almost conclusive of thrombosis. (6) The average duration is about three weeks, and death is generally from pulmonary pyæmia. The chief points in the diagnosis may be gathered from these statements.

Pitt records an interesting case of recovery in a boy of ten, who had otorrhœa for years and was admitted with fever, earache, tenderness, and œdema. A week later he had a rigor, and optic neuritis developed on the right side. The mastoid was explored unsuccessfully. The fever and chills persisting, two days later the lateral sinus was explored. A mass of foul clot was removed and the jugular vein was tied, after which the boy made a satisfactory recovery.

According to Griesinger there is often associated with thrombosis of the lateral sinus venous stasis and painful œdema behind the ear and in the neck. The external jugular vein on the diseased side may be less distended than on the opposite side, since owing to the thrombus in the lateral sinus the internal jugular vein is less full than on the normal side, and the blood from the external jugular can flow more easily into it (Gerhardt).

Treatment.—In marantic individuals roborants and stimulants are indicated. The position assumed in bed should favor both the arterial and venous circulation. The clothing should not restrict the neck, and care should be taken to avoid *bending* of the neck.

The internal administration of potassium iodide and calomel has been

recommended in the autochthonous forms, but no treatment is likely to be of any avail.

The secondary forms, especially those following upon disease of the middle ear, are often amenable to operation, and, especially recently, many lives have been saved by surgical intervention after extensive sinus thrombosis. Macewen's work On Pyogenic Infective Diseases of the Brain and Spinal Cord contains the most exhaustive presentation of the subject of sinus thrombosis and its treatment.

9. Hemiplegia in Children.

Etiology.—Of 135 cases, 60 were in boys and 75 in girls. Right hemiplegia occurred in 79, left in 56. In 15 cases the condition was said to be congenital.

In a great majority the disease sets in during the first or second year; thus of the total number of cases, 95 were under two. Cases above the fifth year are rare, only 10 in my series. Neither alcoholism nor syphilis in the parents appears to play an important *rôle* in this affection. Difficult or abnormal labor is responsible for certain of the cases, particularly injury with the *forceps*. Trauma, such as falls or puncturing wounds, is more rare. The condition followed ligation of the common carotid in one case.

Infectious diseases. All the authors lay special stress upon this factor. In 19 cases in my series the disease came on during or just after one of the specific fevers. I saw one case in which during the height of vaccination convulsions developed, followed by hemiplegia. In a great majority of the cases the disease sets in with a convulsion, in which the child may remain for several hours or longer, and after recovery the paralysis is noticed.

Morbid Anatomy.—In an analysis which I have made of 90 autopsies reported in the literature, the lesions may be grouped under three headings:

(*a*) Embolism, thrombosis, and hæmorrhage, comprising 16 cases, in 7 of which there was blocking of a Sylvian artery, and in 9 hæmorrhage. A striking feature in this group is the advanced age of onset. Ten of the cases occurred in children over six years old.

(*b*) Atrophy and sclerosis, comprising 50 cases. The wasting is either of groups of convolutions, an entire lobe, or the whole hemisphere. The meninges are usually closely adherent over the affected region, though sometimes they look normal. The convolutions are atrophied, firm, and hard, contrasting strongly with the normal gyri. The sclerosis may be diffuse and widespread over a hemisphere, or there may be nodular projections—the hypertrophic sclerosis. Some of the cases show remarkable unilateral atrophy of the hemisphere. In one of my cases the atrophied hemisphere weighed 169 grammes and the normal one 653 grammes. The brain tissue may be a mere shell over a dilated ventricle.

(*c*) Porencephalus, which was present in 24 of the 90 autopsies. This term was applied by Heschel to a loss of substance in the form of cavities

and cysts at the surface of the brain, either opening into and bounded by the arachnoid, and even passing deeply into the hemisphere, or reaching to the ventricle. In the study by Audrey of 103 cases of porencephalus, hemiplegia was mentioned in 68 cases.

Practically, then, in infantile hemiplegia cortical sclerosis and porencephalus are the important anatomical conditions. The primary change in the majority of these cases is still unknown. Porencephalia may result from a defect in development or from hæmorrhage at birth. The etiology is clear in the limited number of cases of hæmorrhage, embolism, and thrombosis, but there remains the large group in which the final change is sclerosis and atrophy. What is the primary lesion in these instances? The clinical history shows that in nearly all these cases the onset is sudden, with convulsions—often with slight fever. Strümpell believes that this condition is due to an inflammation of the gray matter—polio-encephalitis—a view which has not been very widely accepted, as the anatomical proofs are wanting. Gowers suggests that thrombosis may be present in some instances. This might probably account for the final condition of sclerosis, but clinically thrombosis of the veins rarely occurs in healthy children, which appear to be those most frequently attacked by infantile hemiplegia, and post-mortem proof is yet wanting of the association of thrombosis with the disease.

Symptoms.—(*a*) The onset. The disease may set in suddenly without spasms or loss of consciousness. In more than half the cases the child is attacked with partial or general convulsions and loss of consciousness, which may last from a few hours to many days. This is one of the most striking features in the disease. Fever is usually present. The hemiplegia, noticed as the child recovers consciousness, is generally complete. Sometimes the paralysis is not complete at first, but develops after subsequent convulsions. The right side is more frequently affected than the left. The face is commonly not involved.

(*b*) Residual symptoms. In some cases the paralysis gradually disappears and leaves scarcely a trace as the child grows up. The leg, as a rule, recovers more rapidly and more fully than the arm, and the paralysis may be scarcely noticeable. In a majority of cases, however, there is a characteristic hemiplegic gait. The paralysis is most marked in the arm, which is usually wasted; the forearm is flexed at right angles, the hand is flexed, and the fingers are contracted. Motion may be almost completely lost; in other instances the arm can be lifted above the head. Late rigidity, which almost always develops, is the symptom which suggested the name *hemiplegia spastica cerebralis* to Heine, the orthopædic surgeon who first accurately described these cases. It is, however, not constant. The limbs may be quite relaxed even years after the onset. The reflexes are usually increased. In several instances, however, I have known them to be absent. Sensation is, as a rule, not disturbed.

Aphasia is a not uncommon symptom, and occurred in 16 cases of my series—a smaller number than that given in the series of Wallenberg, Gaudard, and Sachs.

Mental Defects.—One of the most serious consequences of infantile

hemiplegia is the failure of mental development. A considerable number of these cases drift into the institutions for feeble-minded children. Three grades may be distinguished—idiocy, which is most common when the hemiplegia has existed from birth; imbecility, which often increases with the development of epilepsy; and feeble-mindedness, a retarded rather than an arrested development.

Epilepsy.—Of the cases in my series, 41 were subjects of convulsive seizures, one of the most distressing sequels of the disease. The seizures may be either transient attacks of *petit mal*, true Jacksonian fits, beginning in and confined to the affected side, or general convulsions.

Post-hemiplegic Movements.—It was in cases of this sort that Weir Mitchell first described the post-hemiplegic movements. They are extremely common, and were present in 34 of my series. There may be either slight tremor in the affected muscles, or incoördinate choreiform movements—the so-called post-hemiplegic chorea—or, lastly,

Athetosis.—In this condition, described by Hammond, there are remarkable spasms of the paralyzed extremities, chiefly of the fingers and toes, and in rare instances of the muscles of the mouth. The movements are involuntary and somewhat rhythmical; in the hand, movements of adduction or abduction and of supination and pronation follow each other in orderly sequence. There may be hyperextension of the fingers, during which they are spread wide apart. This condition is much more frequent in children than in adults. In the latter it may be combined with hemianæsthesia, and the lesion is not cortical, but basic in the neighborhood of the thalamus. The movements are sometimes increased by emotion. They usually persist during sleep.

Treatment.—The possibility of injury to the brain in protracted labor and in forceps cases should be borne in mind by the practitioner. The former entails the greater risk. In infantile hemiplegia the physician at the outset sees a case of ordinary convulsions, perhaps more protracted and severe than usual. These should be checked as rapidly as possible by the use of the bromides, the application of cold or heat, and a brisk purge. During convulsions chloroform may be administered with safety even to the youngest children. When the paralysis is established not much can be hoped from medicines. In only rare instances does the paralysis entirely disappear. When the recovery is partial the "residual paralysis" is similar to that seen in other lesions of the upper motor segment. Thus in the lower extremity it is the flexors of the leg and the dorsal flexors of the foot which are most often permanently paralyzed (Wernicke). The indications are to favor the natural tendency to improve by maintaining the general nutrition of the child, to lessen the rigidity and contractures by massage and passive motion, and if necessary to correct deformities by mechanical or surgical measures. Much may be done by careful manipulation and rubbing and the application of a proper apparatus. In children the aphasia usually disappears. The epilepsy is a distressing and obstinate symptom, for which a cure can rarely be anticipated. Prolonged periods of quiescence are, however, not uncommon. In the Jacksonian fits the bromides rarely do good, unless there is much irritability and excitement.

Operative measures, which have been carried out in several cases, have not, as a rule, been successful. The liability to feeble-mindedness is the most serious outlook in the infantile cerebral palsies. In many cases the damage is irreparable, and idiocy and imbecility result. With patient training and with care many of the children reach a fair measure of intelligence and self-reliance.

IV. TUMORS, INFECTIOUS GRANULOMATA, AND CYSTS OF THE BRAIN.

The following are the most common varieties of new growths within the cranium:

(1) **Infectious Granulomata.**—(*a*) *Tubercle*, which may form large or small growths, usually multiple. Tuberculosis of the glands or bones may be coexistent, but the tuberculous disease of the brain may occur in the absence of other clinically recognizable tuberculous lesions. The disease is most frequent early in life. Three fourths of the cases occur under twenty, and one half of the patients are under ten years of age (Gowers). Of 299 cases of tumor in persons under nineteen collected from various sources by Starr, 152 were tubercle. The nodules are most numerous in the cerebellum and about the base.

(*b*) *Syphiloma* is most commonly found in the hemispheres or about the pons. The tumors are superficial, attached to the arteries or the meninges, and rarely grow to a large size. They may be multiple. The third nerve is particularly prone to syphilitic infiltration, and ptosis is common.

(2) **Tumors.**—(*c*) *Glioma and Neuroglioma.*—These vary greatly in appearance. They may be firm and hard, almost like an area of sclerosis, or soft and very vascular. They persist remarkably for many years. Klebs has called attention to the occurrence of elements in them not unlike ganglion-cells. Tumors of this character may contain the "Spinnen" or spider cells; enormous spindle-shaped cells with single large nuclei; cells like the ganglion-cells of nerve-centres with nuclei and one or more processes; and translucent, band-like fibres, tapering at each end, which result from a vitreous or hyaline transformation of the large spindle-cells. A separate type is also recognizable, in which the cells resemble the ependymal epithelium.

(*d*) *Sarcoma* occurs most commonly in the membranes of the brain and in the pons. It forms some of the largest and most diffusely infiltrating of intracranial growths. Like carcinoma, sarcoma of the brain is usually of very rapid growth.

(*e*) *Carcinoma* not infrequently is secondary to cancer in other parts. It is seldom primary. Occasionally cancerous tumors have been found in symmetrical parts of the brain.

(*f*) Other varieties occur, such as fibroid growths, which usually develop from the membranes; bony tumors, which grow sometimes from the falx, psammoma, and cholesteatoma. Fatty tumors are occasionally found on the corpus callosum.

(3) **Cysts.**—(*g*) These occur between the membranes and the brain, as a result of hæmorrhage or of softening. Porencephalus is a sequel of congenital atrophy or of hæmorrhage, or may be due to a developmental defect. Hydatid cysts have been referred to in the section on parasites. An interesting variety of cyst is that which follows severe injury to the skull in early life.

Symptoms.—(1) **General.**—The following are the most important: *Headache*, either dull, aching, and continuous, or sharp, stabbing, and paroxysmal. It may be diffused over the entire head; sometimes it is limited to the back or front. When in the back of the head it may extend down the neck (especially in tumors in the posterior fossa), and when in the front it may be accompanied with neuralgic pains in the face. Occasionally the pain may be very localized and associated with tenderness on pressure.

Optic neuritis occurs in four fifths of all the cases (Gowers). It is usually double, but occasionally is found in only one eye. A growth may develop slowly and attain considerable size without producing optic neuritis. On the other hand, it may occur with a very small tumor. J. A. Martin, from an extensive analysis of the literature with reference to the localizing value, concludes: When there is a difference in the amount of the neuritis in each eye it is more than twice as probable that the tumor is on the side of the most marked neuritis. It is constant in tumors of the corpora quadrigemina, present in 89 per cent of cerebellar tumors, and absent in nearly two thirds of the cases of tumor of the pons, medulla, and of the corpus callosum. It is least frequent in cases of tuberculous tumor; most common in cases of glioma and cystic tumors.

Vomiting is a common feature, and with headache and optic neuritis makes up the characteristic clinical picture of cerebral tumor. An important point is the absence of definite relation to the meals. A chemical examination shows that the vomiting is independent of digestive disturbances. It may be very obstinate, particularly in growths of the cerebellum and the pons.

Giddiness is often an early symptom. The patient complains of vertigo on rising suddenly or on turning quickly. *Mental Disturbance.*—The patient may act in an odd, unnatural manner, or there may be stupor and heaviness. The patient may become emotional or silly, or symptoms resembling hysteria may develop. *Convulsions*, either general and resembling true epilepsy or localized (Jacksonian) in character. There may be *slowing of the pulse*, as in all cases of increased intracranial pressure.

(2) **Localizing Symptoms.**—Focal symptoms often occur, but it must not be forgotten that these may be *indirectly* produced. The smaller the tumor and the less marked the general symptoms of cerebral compression, the more likely is it that any focal symptoms occurring are of *direct* origin.

(*a*) *Central Motor Area.*—The symptoms are either irritative or destructive in character. Irritation in the lower third may produce spasm in the muscles of the face, in the angle of the mouth, or in the tongue. The spasm with tingling may be strictly limited to one muscle group before extending to others, and this Seguin terms the *signal symptom*. The middle third of the motor area contains the centres controlling the arm, and here,

too, the spasm may begin in the fingers, in the thumb, in the muscles of the wrist, or in the shoulder. In the upper third of the motor areas the irritation may produce spasm beginning in the toes, in the ankles, or in the muscles of the leg. In many instances the patient can determine accurately the point of origin of the spasm, and there are important sensory disturbances, such as numbness and tingling, which may be felt first at the region affected.

In all cases it is important to determine, first, the point of origin, the *signal symptom;* second, the order or march of the spasm; and third, the subsequent condition of the parts first affected, whether it is a state of paresis or anæsthesia.

Destructive lesions in the motor zone cause paralysis, which is often preceded by local convulsive seizures; there may be a monoplegia, as of the leg, and convulsive seizures in the arm, often due to irritation in these centres. Tumors in the neighborhood of the motor area may cause localized spasms and subsequently, as the centres are invaded by the growth, paralysis occurs. On the left side, growths in the third frontal or Broca's convolution may cause motor aphasia.

(*b*) *Prefrontal Region.*—Neither motor nor sensory disturbance may be present. The general symptoms are often well marked. The most striking feature of growths in this region is mental torpor and gradual imbecility. In its extension downward the tumor may involve on the left side the lower frontal convolution and produce aphasia, or in its progress backward cause irritative or destructive lesions of the motor area. Exophthalmos on the side of the tumor may occur and be helpful in diagnosis, as in the case reported by Thomas and Keene.

(*c*) Tumors in the *parieto-occipital lobe* may grow to a large size without causing any symptoms. There may be word-blindness and mind-blindness when the angular gyrus and its underlying white matter is involved, and paraphasia.

(*d*) Tumors of the *occipital lobe* produce hemianopia, and a bilateral lesion may produce blindness. Tumors in this region on the left hemisphere may be associated with word-blindness and mind-blindness.

(*e*) Tumors in the *temporal lobe* may attain a large size without producing symptoms. In their growth they involve the lower motor centres. On the left side involvement of the first gyrus and the transverse temporal gyri (auditory sense area) may be associated with word-deafness.

(*f*) Tumors growing in the neighborhood of the *basal ganglia* produce hemiplegia from involvement of the internal capsule. Limited growths in either the nucleus caudatus or the nucleus lentiformis of the corpus striatum do not necessarily cause paralysis. Tumors in the thalamus opticus may also, when small, cause no symptoms, but increasing they may involve the fibres of the sensory portion of the internal capsule, producing hemianopia and sometimes hemianæsthesia. Growths in this situation are apt to cause early optic neuritis, and, growing into the third ventricle, may cause a distention of the lateral ventricles. In fact, pressure symptoms from this cause and paralysis due to involvement of the internal capsule are the chief symptoms of tumor in and about these ganglia. If the ventrolateral group

of nuclei in the thalamus be involved there may be unilateral disturbances of cutaneous and muscular sense, hemichorea, or movement ataxia.

Growths in the *corpora quadrigemina* are rarely limited, but most commonly involve the crura cerebri as well. Ocular symptoms are marked. The pupil reflex is lost and there is nystagmus. In the gradual growth the third nerve is involved as it passes through the crus, in which case there will be oculo-motor paralysis on one side and hemiplegia on the other, a combination almost characteristic of unilateral disease of the crus.

(*g*) Tumors of the *pons* and *medulla*. The symptoms are chiefly those of pressure upon the nerves emerging in this region. In disease of the pons the nerves may be involved alone or with the pyramidal tract. Of 52 cases analyzed by Mary Putnam Jacobi, there were 13 in which the cerebral nerves were involved alone, 13 in which the limbs were affected, and 26 in which there was hemiplegia and involvement of the nerves. Twenty-two of the latter had what is known as alternate paralysis—i. e., involvement of the nerves on one side and of the limbs on the opposite side. In 4 cases there were no motor symptoms. In tuberculosis (or syphilis) a growth at the inferior and inner aspects of the crus may cause paralysis of the third nerve on one side, and of the face, tongue, and limbs on the opposite side (syndrome of Weber). A tumor growing in the lower part of the pons usually involves the sixth nerve, producing internal strabismus; the seventh nerve, producing facial paralysis; and the auditory nerve, causing deafness. Conjugate deviation of the eyes to the side opposite that on which there is facial paralysis also occurs. When the motor cerebral nerves are involved the paralyses are of the peripheral type (lower segment paralyses).

Tumors of the *medulla* may involve the cerebral nerves alone or cause in some instances a combination of hemiplegia with paralysis of the nerves. Paralyses of the nerves are helpful in topical diagnosis, but the fact must not be overlooked that one or more of the cerebral nerves may be paralyzed as a result of a much increased general intracranial pressure. Signs of irritation in the ninth, tenth, and eleventh nerves are usually present, and produce difficulty in swallowing, irregular action of the heart, irregular respiration, vomiting, and sometimes retraction of the head and neck. The hypoglossal nerve is least often affected. The gait may be unsteady or, if there is pressure on the cerebellum, ataxic. Occasionally there are sensory symptoms, numbness, and tingling. Toward the end convulsions may occur.

Diagnosis.—From the general symptoms alone the existence of tumor may be determined, for the combination of headache, optic neuritis, and vomiting is distinctive. A gradual increase in the intensity of the symptoms is usually seen. It must not be forgotten that severe headache and neuro-retinitis may be caused by Bright's disease. The localization must be gathered from the consideration of the symptoms above detailed and from the data given in the section on Topical Diagnosis of Diseases of the Brain. Mistakes are most likely to occur in connection with uræmia, hysteria, and general paralysis; but careful consideration of all the circumstances of the case usually enables the practitioner to avoid error. Auscultatory percussion is occasionally of service in localization.

Prognosis.—Syphilitic tumors alone are amenable to medical treatment. Tuberculous growths occasionally cease to grow and become calcified. The gliomata and fibromata, particularly when the latter grow from the membranes, may last for years. I have described a case of small, hard glioma, in which the Jacksonian epilepsy persisted for fourteen years. Hughlings Jackson has reported cases of glioma in which the symptoms lasted for over ten years. The more rapidly growing sarcomata usually prove fatal in from six to eighteen months. Death may be sudden, particularly in growths near the medulla; more commonly it is due to coma in consequence of gradual increase in the intracranial pressure.

Treatment.—(*a*) *Medical.*—If there is a suspicion of syphilis the iodide of potassium and mercury should be given. Nowhere do we see more brilliant therapeutical effects than in certain cases of cerebral gummata. The iodide should be given in increasing doses. In tuberculous tumors the outlook is less favorable, though instances of cure are reported, and there is post-mortem evidence to show that the solitary tuberculous tumors may undergo changes and become obsolete. A general tonic treatment is indicated in these cases. The headache usually demands prompt treatment. The iodide of potassium in full doses sometimes gives marked relief. An ice-cap for the head or, in the occipital headache, the application of the Paquelin cautery may be tried. The bromides are not of much use in the headache from this cause, and, as the last resort, morphia must be given. For the convulsions bromide of potassium is of little service.

(*b*) *Surgical.*—Tumors of the brain have been successfully removed by Macewen, Horsley, Keen, and others. The number of cases for operation, however, is small. Four fifths at least of all the cases are probably unsuitable, or of such a nature as to render an operation fatal. The most advantageous cases are the localized fibromata growing from the dura and only compressing the brain substance, as in Keen's remarkable case. The safety with which the exploratory operation can be made warrants it in all doubtful cases.

V. INFLAMMATION OF THE BRAIN.

1. Acute Encephalitis.

A focal or diffuse inflammation of the brain substance, usually of the gray matter (poliencephalitis), is met with (*a*) as a result of trauma; (*b*) in certain intoxications, alcohol, food poisoning, and gas poisoning; and (*c*) following the acute infections. The anatomical features are those of an acute hæmorrhagic poliencephalitis, corresponding in histological details with acute polio-myelitis. Focal forms are seen in ulcerative endocarditis, in which the gray matter may present deeply hæmorrhagic areas, firmer than the surrounding tissue. In the fevers there may be more extensive regions, involving two or three convolutions. This acute hæmorrhagic poliencephalitis superior is thought by Strümpell to be the essential lesion in infantile hemiplegia. Localizing symptoms are usually present, though

they may be obscured in the severity of the general infection. The most typical encephalitis accompanies the meningitis in cerebro-spinal fever.

In acute mania, in delirium tremens, in chorea insaniens, in the maniacal form of exophthalmic goitre, and in the so-called cerebral forms of the malignant fevers the gray cortex is deeply congested, moist, and swollen, and with the recent finer methods of research will probably show changes which may be classed as encephalitis.

The *symptoms* are not very definite. In severe forms they are those of an acute infection; some cases have been mistaken for typhoid fever. The onset may be abrupt in an individual apparently healthy. Other cases have occurred in the convalescence from the fevers, particularly influenza. One of J. J. Putnam's cases followed mumps. The general symptoms are those which accompany all severe acute affections of the brain—headache, somnolence, coma, delirium, vomiting, etc. The local symptoms are very varied, depending on the extent of the lesions, and may be irritative or paralytic. Usually fatal within a few weeks, cases may drag on for weeks or months and recover.

2. Abscess of the Brain.

Etiology.—Suppuration of the brain substance is rarely if ever primary, but results, as a rule, from extension of inflammation from neighboring parts or infection from a distance through the blood. The question of idiopathic brain abscess need scarcely be considered, though occasionally instances occur in which it is extremely difficult to assign a cause. There are three important etiological factors:

(1) Trauma. Falls upon the head or blows, with or without abrasion of the skin. More commonly it follows fracture or punctured wounds. In this group meningitis is frequently associated with the abscess.

(2) By far the most important infective foci are those which arise in direct extension from disease of the middle ear or of the mastoid cells. From the roof of the mastoid antrum the infection readily passes to the sigmoid sinus and induces an infective thrombosis. In other instances the dura becomes involved, and a sub-dural abscess is formed, which may readily involve the arachnoid or the pia mater. In another group the inflammation extends along the lymph spaces, or the thrombosed veins, into the substance of the brain and causes suppuration. Macewen thinks that without local areas of meningitis the infective agents may be carried through the lymph and blood channels into the cerebral substance. Infection which extends from the roof of the mastoid process is most likely to be followed by abscess in the temporal lobe, while infection extending from the posterior wall causes most frequently sinus thrombosis and cerebellar abscess.

(3) In septic processes. Abscess of the brain is not often found in pyæmia. In ulcerative endocarditis multiple foci of suppuration are common. Localized bone-disease and suppuration in the liver are occasional causes. Certain inflammations in the lungs, particularly bronchiectasis, which was present in 17 of 38 cases of these so-called "pulmonal cerebral

abscesses" collected by R. T. Williamson, are liable to be followed by abscess. It is an occasional complication of empyema. Abscess of the brain may follow the specific fevers. Bristowe has called attention to its occurrence as a sequel of influenza. The largest number of cases occur between the twentieth and fortieth years, and the condition is more frequent in men than in women. Holt has collected 25 cases in children under five years of age, the chief causes of which were otitis media and trauma.

Morbid Anatomy.—The abscess may be solitary or multiple, diffuse or circumscribed. Practically any one of the different varieties of pyogenic bacteria may be concerned. The bacteriological examination often shows a mixture of different varieties. Occasionally cultures are sterile, owing to death of the bacteria. In the acute, rapidly fatal cases following injury the suppuration is not limited; but in long-standing cases the abscess is enclosed in a definite capsule, which may have a thickness of from 2 to 5 mm. The pus varies much in appearance, depending upon the age of the abscess. In early cases it may be mixed with reddish *débris* and softened brain matter, but in the solitary encapsulated abscess the pus is distinctive, having a greenish tint, an acid reaction, and a peculiar odor, sometimes like that of sulphuretted hydrogen. The brain substance surrounding the abscess is usually œdematous and infiltrated. The size varies from that of a walnut to that of a large orange. There are cases on record in which the cavity has occupied the greater portion of a hemisphere. Multiple abscesses are usually small. In four fifths of all cases the abscess is solitary. Suppuration occurs most frequently in the cerebrum, and the temporal lobe is more often involved than other parts. The cerebellum is the next most common seat, particularly in connection with ear-disease.

Symptoms.—Following injury or operation the disease may run an *acute* course, with fever, headache, delirium, vomiting, and rigors. The symptoms are those of an acute meningo-encephalitis, and it may be very difficult to determine, unless there are localizing symptoms, whether there is really suppuration in the brain substance. In the cases following ear disease the symptoms may at first be those of meningeal irritation. There may be irritability, restlessness, severe headache, and aggravated earache. Other striking symptoms, particularly in the more prolonged cases, are drowsiness, slow cerebration, vomiting, and optic neuritis. In the chronic form of brain abscess which may follow injury, otorrhœa, or local lung trouble, there may be a latent period ranging from one or two weeks to several months, or even a year or more. In the "silent" regions, when the abscess becomes encapsulated there may be no symptoms whatever during the latent period. During all this time the patient may be under careful observation and no suspicion be aroused of the existence of suppuration. Then severe headache, vomiting, fever, set in, perhaps with a chill. So, too, after a blow upon the head or a fracture the symptoms of the lesion may be transient, and months afterward cerebral symptoms of the most aggravated character may develop.

The localization of the lesion is often difficult. In or near the motor region there may be convulsions or paralysis, and it is to be remembered that an abscess in the temporal lobe may compress the lower motor centres

and produce paralysis of the arm and face and on the left side cause aphasia. A large abscess may exist in the frontal lobe without causing paralysis, but in these cases there is almost always some mental dulness. In the temporal lobe, the common seat, there may be no focalizing symptoms. So also in the parieto-occipital region; though here early examination may lead to the detection of hemianopia. In abscess of the cerebellum vomiting is common. If the middle lobe is affected there may be staggering—cerebellar incoördination. Localizing symptoms in the pons and other parts are still more uncertain.

Diagnosis.—In the acute cases there is rarely any doubt. A consideration of possible etiological factors is of the highest importance. The history of injury followed by fever, marked cerebral symptoms, the development of rigors, delirium, and perhaps paralysis, make the diagnosis certain. In chronic ear-disease, such cerebral symptoms as drowsiness and torpor, with irregular fever, supervening upon the cessation of a discharge, should excite the suspicion of abscess. Cases in which suppurative processes exist in the orbit, nose, or naso-pharynx, or in which there has been subcutaneous phlegmon of the head or neck, a parotitis, a facial erysipelas, or tuberculous or syphilitic disease of the bones of the skull, should be carefully watched, and immediately investigated should cerebral symptoms appear. It is particularly in the chronic cases that difficulties arise. The symptoms resemble those of tumor of the brain; indeed, they are those of tumor plus fever. Choked disk, however, so commonly associated with tumor, is very frequently absent in abscess of the brain. In a patient with a history of trauma or with localized lung or pleural trouble, who for weeks or months has had slight headache or dizziness, the onset of a rapid fever, especially if it be intermittent and associated with rigors, intense headache, and vomiting, point strongly to abscess. The pulse-rate in cases of cerebral abscess is usually accelerated, but cases are not rare in which it is slowed. Macewen lays stress upon the value of percussion of the skull as an aid in diagnosis. The note, which is uniformly dull, becomes much more resonant when the lateral ventricles are distended in cerebellar abscess and in conditions in which the venæ Galeni are compressed.

It is not always easy to determine whether the meninges are involved with the abscess. Often in ear-disease the condition is that of meningo-encephalitis. Sometimes in association with acute ear-disease the symptoms may simulate closely cerebral meningitis or even abscess. Indeed, Gowers states that not only may these general symptoms be produced by ear-disease, but even distinct optic neuritis.

Treatment.—A remarkable advance has been made of late years in dealing with these cases, owing to the impunity with which the brain can be explored. In ear-disease free discharge of the inflammatory products should be promoted and careful disinfection practised. The treatment of injuries and fractures comes within the scope of the surgeon. The acute symptoms, such as fever, headache, and delirium, must be treated by rest, an ice-cap, and, if necessary, local depletion. In all cases, when a reasonable suspicion exists of the occurrence of abscess, the trephine should be used and the brain explored. The cases following ear-disease, in which

the suppuration is in the temporal lobe or in the cerebellum, offer the most favorable chances of recovery. The localization can rarely be made accurately in these cases, and the operator must be guided more by general anatomical and pathological knowledge. In cases of injury the trephine should be applied over the seat of the blow or the fracture. In ear-disease the suppuration is most frequent in the temporal lobe or in the cerebellum, and the operation should be performed at the points most accessible to these regions. And, lastly, a most important, one might almost say essential, factor in the successful treatment of intracranial suppuration is an intelligent knowledge on the part of the surgeon of the work and works of William Macewen.

VI. HYDROCEPHALUS.

Definition.—A condition, congenital or acquired, in which there is a great accumulation of fluid within the ventricles of the brain.

The term hydrocephalus has also been applied to the collection of fluid between the cortex of the brain and the skull, known in this situation as *h. externus* or *h. ex vacuo*, a condition common in cases of atrophy of the brain substance, met with in old age, after hæmorrhages, softenings, or scleroses, in lingering and cachectic diseases, as cancer, chronic nephritis, chronic alcoholism, and sometimes in rickets. Occasionally the disease is caused by meningeal cysts. A true dropsy, however, of the arachnoid sac probably does not occur.

The cases may be divided into three groups—idiopathic internal hydrocephalus (serous meningitis), congenital or infantile, and secondary or acquired.

(1) **Serous Meningitis** (Quincke) (*Idiopathic Internal Hydrocephalus; Angio-neurotic Hydrocephalus*).—This remarkable form, described by Quincke, is very important, since a knowledge of the condition may explain very anomalous and puzzling cases. It is an ependymitis causing a serous effusion into the ventricles, with distention and pressure effects. It may be compared to the serous exudates in the pleura or in synovial membranes. It is not certain that the process is inflammatory, and Quincke likens it to the angio-neurotic œdema of the skin. In very acute cases the ependyma may be smooth and natural looking; in more chronic cases it may be thickened and sodden. The exudate does not differ from the normal, and if on lumbar puncture a fluid is removed of a specific gravity above 1.009, with albumin above two per one thousand, the condition is more likely to be hydrocephalus from stasis, secondary to tumor, etc.

Both children and adults are affected, the latter more frequently. In the acute form the condition is mistaken for tuberculous or purulent meningitis. There are headache, retraction of the neck, and signs of increased intracranial pressure, choked disks, slow pulse, etc. Fever is usually absent, but I have seen one case with recurring paroxysms of fever, and Morton Prince has described a similar one. In both the exudate was clear and the ependyma not acutely inflamed. Quincke has reported cases of recovery. In the chronic form the symptoms are those of tumor—general, such as

headache, slight fever, somnolence, and delirium; and local, as exophthalmos, optic neuritis, spasms, and rigidity of muscles and paralysis of the cerebral nerves. Remarkable exacerbations occur, and the symptoms vary in intensity from day to day. Recovery may follow after an illness of many weeks, and some of the reported cases of disappearance of all symptoms of brain tumor belong in this category.

(2) **Congenital Hydrocephalus.**—The enlarged head may obstruct labor; more frequently the condition is noticed some time after birth. The cause is unknown. It has occurred in several members of the same family.

The anatomical condition in these cases offers no clew to the nature of the trouble. The lateral ventricles are enormously distended, but the ependyma is usually clear, sometimes a little thickened and granular, and the veins large. The choroid plexuses are vascular, sometimes sclerotic, but often natural looking. The third ventricle is enlarged, the aqueduct of Sylvius dilated, and the fourth ventricle may be distended. The quantity of fluid may reach several litres. It is limpid and contains a trace of albumin and salts. The changes in consequence of this enormous ventricular distention are remarkable. The cerebral cortex is greatly stretched, and over the middle region the thickness may amount to no more than a few millimetres without a trace of the sulci or convolutions. The basal ganglia are flattened. The skull enlarges, and the circumference of the head of a child of three or four years may reach 25 or even 30 inches. The sutures widen, Wormian bones develop in them, and the bones of the cranium become exceedingly thin. The veins are marked beneath the skin. A fluctuation wave may sometimes be obtained, and Fisher's brain murmur may be heard. The orbital plates of the frontal bone are depressed, causing exophthalmos, so that the eyeballs cannot be covered by the eyelids. The small size of the face, widening somewhat above, is striking in comparison with the enormously expanded skull.

Convulsions may occur. The reflexes are increased, the child learns to walk late, and ultimately in severe cases the legs become feeble and sometimes spastic. Sensation is much less affected than motility. Choked disk is not uncommon. The mental condition is variable; the child may be bright, but, as a rule, there is some grade of imbecility. The congenital cases usually die within the first four or five years. The process may be arrested and the patient may reach adult life. Cases of this sort are not very uncommon. Even when extreme, the mental faculties may be retained, as in Bright's celebrated patient, Cardinal, who lived to the age of twenty-nine, and whose head was translucent when the sun was shining behind him. Care must be taken not to mistake the rachitic head for hydrocephalus.

(3) **Acquired Chronic Hydrocephalus.**—This is stated to be occasionally primary (idiopathic)—that is to say, it comes on spontaneously in the adult without observable lesion. Dean Swift is said to have died of hydrocephalus, but this seems very unlikely. It is based upon the statement that "he (Mr. Whiteway) opened the skull and found much water in the brain," a condition no doubt of *h. ex vacuo*, due to the wasting associated with his prolonged illness and paralysis. In nearly all cases there is either

a tumor at the base of the brain or in the third ventricle, which compresses the venæ Galeni. The passage from the third to the fourth ventricle may be closed, either by a tumor or by parasites. More rarely the foramen of Magendie, through which the ventricles communicate with the cerebrospinal meninges, becomes closed by meningitis. These conditions, occurring in adults, may produce the most extreme hydrocephalus without any enlargement of the head. Even when the tumor begins early in life there may be no expansion of the skull. In the case of a girl aged sixteen, blind from her third year, the head was not unusually large, the ventricles were enormously distended, and in the Rolandic region the brain substance was only 5 mm. in thickness. A tumor occupied the third ventricle. In a case of cholesteatoma of the floor of the third ventricle, in which the symptoms persisted at intervals for eight or nine years, the ventricles were enormously distended without enlargement of the skull. In other instances the sutures separate and the head gradually enlarges.

The symptoms of hydrocephalus in the adult are curiously variable. In the first case mentioned there were early headaches and gradual blindness; then a prolonged period in which she was able to attend to her studies. Headaches again supervened, the gait became irregular and somewhat ataxic. Death occurred suddenly. In the other case there were prolonged attacks of coma with a slow pulse, and on one occasion the patient remained unconscious for more than three months. Gradually progressing optic neuritis without focalizing symptoms, headache, and attacks of somnolence or coma are suggestive symptoms. These cases of acquired chronic hydrocephalus cannot be certainly diagnosed during life, though in certain instances the condition may be suspected.

Treatment.—Very little can be done to relieve hydrocephalus. Medicines are powerless to cause the absorption of the fluid. More rational is the system of gradual compression, with or without the withdrawal of small quantities of the fluid. The compression may be made by means of broad plasters, so applied as to cross each other on the vertex, and another may be placed round the circumference. In the meningitis serosa Quincke advises the use of mercury.

Of late years puncture of the ventricles, an operation which has been abandoned, has been revived; it has been resorted to in the meningitis serosa. When pressure symptoms are marked Quincke's procedure may be used. He recommends puncture of the subarachnoid sac between the third and the fourth lumbar vertebræ. At this point the spinal cord cannot be touched. The advantages are a slower removal of fluid and less danger of collapse.

VI. DISEASES OF THE PERIPHERAL NERVES.

I. NEURITIS (*Inflammation of the Bundles of Nerve Fibres*).

Neuritis may be *localized* in a single nerve, or *general*, involving a large number of nerves, in which case it is usually known as *multiple neuritis* or *polyneuritis*.

Etiology.—*Localized neuritis* arises from (*a*) cold, which is a very frequent cause, as, for example, in the facial nerve. This is sometimes known as rheumatic neuritis. (*b*) Traumatism—wounds, blows, direct pressure on the nerves, the tearing and stretching which follow a dislocation or a fracture, and the hypodermic injection of ether. Under this section come also the professional palsies, due to pressure in the exercise of certain occupations. (*c*) Extension of inflammation from neighboring parts, as in a neuritis of the facial nerve due to caries in the temporal bone, or in that met with in syphilitic disease of the bones, disease of the joints, and occasionally in tumors.

Multiple neuritis has a very complex etiology, the causes of which may be classified as follows: (*a*) The poisons of infectious diseases, as in leprosy, diphtheria, typhoid fever, small-pox, scarlet fever, and occasionally in other forms; (*b*) the organic poisons, comprising the diffusible stimulants, such as alcohol and ether, bisulphide of carbon and naphtha, and the metallic bodies, such as lead, arsenic, and mercury; (*c*) cachectic conditions, such as occur in anæmia, cancer, tuberculosis, or marasmus from any cause; (*d*) the endemic neuritis or beri-beri; and (*e*) lastly, there are cases in which none of these factors prevail, but the disease sets in suddenly after overexertion or exposure to cold.

Morbid Anatomy.—In neuritis due to the extension of inflammation the nerve is usually swollen, infiltrated, and red in color. The inflammation may be chiefly perineural or it may pass into the deeper portion—*interstitial* neuritis—in which form there is an accumulation of lymphoid elements between the nerve bundles. The nerve fibres themselves may not appear involved, but there is an increase in the nuclei of the sheath of Schwann. The myelin is fragmented, the nuclei of the internodal cells are swollen, and the axis cylinders present varicosities or undergo granular degeneration. Ultimately the nerve fibres may be completely destroyed and replaced by a fibrous connective tissue in which much fat is sometimes deposited—the *lipomatous neuritis* of Leyden.

In other instances the condition is termed *parenchymatous* neuritis, in which the changes are like those met with in the secondary or Wallerian degeneration, which follows when the nerve fibre is cut off from the cell body of the neurone to which it belongs. The medullary substance and the axis cylinders are chiefly involved, the interstitial tissue being but little altered or only affected secondarily. The myelin becomes segmented and divides into small globules and granules, and the axis cylinders become granular, broken, subdivided, and ultimately disappear. The nuclei of the sheath of Schwann proliferate and ultimately the fibres are reduced to a

state of atrophic tubes without a trace of the normal structure. The muscles connected with the degenerated nerves usually show marked atrophic changes, and in some instances the change in the nerve sheath appears to extend directly to the interstitial tissue of the muscles—the *neuritis fascians* of Eichhorst.

Symptoms.—(*a*) **Localized Neuritis.**—As a rule the constitutional disturbances are slight. The most important symptom is pain of a boring or stabbing character, usually felt in the course of the nerve and in the parts to which it is distributed. The nerve itself is sensitive to pressure, probably, as Weir Mitchell suggests, owing to the irritation of its nervi nervorum. The skin may be slightly reddened or even œdematous over the seat of the inflammation. Mitchell has described increase in the temperature and sweating in the affected region, and such trophic disturbances as effusion into the joints and herpes. The function of the muscle to which the nerve fibres are distributed is impaired, motion is painful, and there may be twitchings or contractions. The tactile sensation of the part may be somewhat deadened, even when the pain is greatly increased. In the more chronic cases of local neuritis, such, for instance, as follow the dislocation of the humerus, the localized pain, which at first may be severe, gradually disappears, though some sensitiveness of the brachial plexus may persist for a long time, and the nerve cords may be felt to be swollen and firm. The pain is variable—sometimes intense and distressing; at others not causing much inconvenience. Numbness and formication may be present and the tactile sensation may be greatly impaired. The motor disturbances are marked. Ultimately there is extreme atrophy of the muscles. Contractures may occur in the fingers. The skin may be reddened or glossy, the subcutaneous tissue œdematous, and the nutrition of the nails may be defective. In the rheumatic neuritis subcutaneous fibroid nodules may develop.

A neuritis limited at first to a peripheral nerve may extend upward—the so-called ascending or migratory neuritis—and involve the larger nerve trunks, or even reach the spinal cord, causing subacute myelitis (Gowers). The condition is rarely seen in the neuritis from cold, or in that which follows fevers; but it occurs most frequently in traumatic neuritis. J. K. Mitchell, in his monograph On Injuries of Nerves (1895), concludes that the larger nerve trunks are most susceptible, and that the neuritis may spread either up or down, the former being the most common. The paralysis secondary to visceral disease, as of the bladder, may be due to an ascending neuritis. The inflammation may extend to the nerves of the other side, either through the spinal cord or its membranes, or without any involvement of the nerve centres, the so-called sympathetic neuritis. The electrical changes in localized neuritis vary a great deal, depending upon the extent to which the nerve is injured. The lesion may be so slight that the nerve and the muscles to which it is distributed may react normally to both currents; or it may be so severe that the typical reaction of degeneration develops within a few days—i. e., the nerve does not respond to stimulation by either current, while the muscle reacts only to the galvanic current and in a peculiar manner. The contraction caused is slow and lazy, instead

of sharp and quick as in the normal muscle, and the AnC contraction is usually stronger than the CC contraction. Between these two extremes there are many different grades, and a careful electrical examination is most important as an aid to diagnosis and prognosis.*

The duration varies from a few days to weeks or months. A slight traumatic neuritis may pass off in a day or two, while the severer cases, such as follow unreduced dislocation of the humerus, may persist for months or never be completely relieved.

(*b*) **Multiple Neuritis.**—This presents a complex symptomatology. The following are the most important groups of cases:

(1) *Acute Febrile Polyneuritis.*—The attack follows exposure to cold or overexertion, or, in some instances, comes on spontaneously. The onset resembles that of an acute infectious disease. There may be a definite chill, pains in the back and limbs or joints, so that the case may be thought to be acute rheumatism. The temperature rises rapidly and may reach 103° or 104°. There are headache, loss of appetite, and the general symptoms of acute infection. The limbs and back ache. Intense pain in the nerves, however, is by no means constant. Tingling and formication are felt in the fingers and toes, and there is increased sensitiveness of the nerve trunks or of the entire limb. Loss of muscular power, first marked, perhaps, in the legs, gradually comes on and extends with the features of an ascending paralysis. In other cases the paralysis begins in the arms. The extensors of the wrists and the flexors of the ankles are early affected, so that there is foot and wrist drop. In severe cases there is general loss of muscular power, producing a flabby paralysis, which may extend to the muscles of the face and to the intercostals, and respiration may be carried on by the diaphragm alone. The muscles soften and waste rapidly. There may be only hyperæsthesia with soreness and stiffness of the limbs; in some cases, increased sensitiveness with anæsthesia; in other instances the sensory disturbances are slight. The clinical picture is not to be distinguished, in many cases, from Landry's paralysis; in others, from the subacute myelitis of Duchenne.

The course is variable. In the most intense forms the patient may die in a week or ten days, with involvement of the respiratory muscles or from paralysis of the heart. As a rule in cases of moderate severity, after persisting for five or six weeks, the condition remains stationary and then slow improvement begins. The paralysis in some muscles may persist for many months and contractures may occur from shortening of the muscles, but even when this occurs the outlook is, as a rule, good, although the paralysis may have lasted for a year or more.

(2) *Recurring Multiple Neuritis.*—Under the term *polyneuritis recurrens* Mary Sherwood has described from Eichhorst's clinic 2 cases in adults—in one case involving the nerves of the right arm, in the other both legs. In one patient there were three attacks, in the other two, the distribution in the various attacks being identical. The subject has recently been fully discussed by H. M. Thomas (Phila. Med. Jour., 1898, i).

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* See under Facial Paralysis.

(3) *Alcoholic Neuritis.*—This, perhaps the most important form of multiple neuritis, was graphically described in 1822 by James Jackson, Sr., of Boston. Wilks recognized it as alcoholic paraplegia, but the starting-point of the recent researches on the disease dates from the observations of Dumenil, of Rouen. Of late years our knowledge of the disease has extended rapidly, owing to the researches of Huss, Leyden, James Ross, Buzzard, and Henry Hun. It occurs most frequently in women, particularly steady, quiet tipplers. Its appearance may be the first revelation to the physician or to the family of habits of secret drinking. The onset is usually gradual, and may be preceded for weeks or months by neuralgic pains and tingling in the feet and hands. Convulsions are not uncommon. Fever is rare. The paralysis gradually sets in, at first in the feet and legs, and then in the hands and forearms. The extensors are affected more than the flexors, so that there is wrist-drop and foot-drop. The paralysis may be thus limited and not extend higher in the limbs. In other instances there is paraplegia alone, while in the most extreme cases all the extremities are involved. In rare instances the facial muscles and the sphincters are also affected. The sensory symptoms are very variable. There are cases in which there are numbness and tingling only, without great pain. In other cases there are severe burning or boring pains, the nerve trunks are sensitive, and the muscles are sore when grasped. The hands and feet are frequently swollen and congested, particularly when held down for a few moments. The cutaneous reflexes as a rule are preserved. The deep reflexes are usually lost.

The course of these alcoholic cases is, as a rule, favorable, and after persisting for weeks or months improvement gradually begins, the muscles regain their power, and even in the most desperate cases recovery may follow. The extensors of the feet may remain paralyzed for some time, and give to the patient a distinctive walk, the so-called *steppage* gait, characteristic of peripheral neuritis. It is sometimes known as the pseudo-tabetic gait, although in reality it could not well be mistaken for the gait of ataxia. The foot is thrown forcibly forward, the toe lifted high in the air so as not to trip upon it. The heel is brought down first and then the entire foot. It is an awkward, clumsy gait, and gives the patient the appearance of constantly stepping over obstacles. Among the most striking features of alcoholic neuritis are the mental symptoms. Delirium is common, and there may be hallucinations with extravagant ideas, resembling somewhat those of general paralysis. In some cases the picture is that of ordinary delirium tremens, but the most peculiar and almost characteristic mental disorder is that so well described by Wilks, in which the patient loses all appreciation of time and place, and describes with circumstantial details long journeys which, he says, he has recently taken, or tells of persons whom he has just seen.

(4) *Multiple Neuritis in the Infectious Diseases.*—This has been already referred to, particularly in diphtheria, in which it is most common. The peripheral nature of the lesion in these instances has been shown by post-mortem examination. The outlook is usually favorable and, except in diphtheria, fatal cases are uncommon. Multiple neuritis in tuberculosis, dia-

betes, and syphilis is of the same nature, being probably due to toxic materials absorbed into the blood.

(5) *Arsenical and Saturnine Neuritis.*—The arsenical neuritis is not common; only a single instance of it has come under my observation. Only one case to my knowledge has followed the use of Fowler's solution in my ward or dispensary practice, although I am in the habit of giving in chorea and anæmia doses which might be regarded as excessive. The most common causes are accidental poisoning, as in the cases reported by Mills. In a case of E. G. Cutler the patient got the arsenic from green-paper tags, which he was in the habit of putting in his mouth. The general symptoms are not unlike those of alcoholic paralysis; the weakness of the extensors is marked and the *steppage* gait characteristic. The neuritis due to lead has been discussed in the consideration of lead poisoning. The special involvement of the motor nerves and the great frequency of the occurrence of wrist-drop are the peculiarities of this form. The changes in the cell bodies of the neurones in cases of poisoning with lead and arsenic have recently been studied by Lugaro by means of the method of Nissl.

A similar form of neuritis is caused by the bisulphide of carbon and by the protracted use of tea (M. A. Starr).

(6) *Endemic Neuritis, Beri-beri,* has been considered under the Infectious Diseases.

Anæsthesia Paralysis.—Here perhaps may most appropriately be considered the forms of paralysis following the use of anæsthetics. Much has been written in the past few years upon this subject, which has been very fully considered by Garrigues (American Journal of the Medical Sciences, 1897, i). There are two groups of cases:

1. Pressure paralysis, in which, owing to the position, the nerves have been compressed, either the humerus against the brachial plexus or the musculo-spiral against the table. The pressure most frequently occurs when the arm is elevated alongside the head, as in laparotomy done in the Trendelenberg position, or held out from the body, as in breast amputations. Instances of paralysis of the crural by Robb's leg-holder are also reported.

2. Paralysis from cerebral lesions during etherization. In one of Garrigues' cases paralysis followed the operation, and at the autopsy, seven weeks later, softening of the brain was found. Apoplexy or embolism may develop during the anæsthesia. In Montreal a cataract operation was performed on an old man. He did not recover from the anæsthetic; I found post mortem a cerebral hæmorrhage. A man was admitted to the Philadelphia Hospital on the 26th completely comatose; the day previously ether had been given for a minor operation. He never recovered consciousness, but remained deeply comatose, with great muscular relaxation, low temperature, 97.5°, and noisy respirations; he died on the 28th. There was, unfortunately, no autopsy. Epileptic convolutions may occur during the anæsthesia, and may even prove fatal. The possibility has to be considered of paralysis from loss of blood in prolonged operations, though I have no personal knowledge of any such cases.

And, lastly, a paralysis might result from the toxic affects of the ether in a very protracted administration.

Diagnosis.—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion.

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are therefore important symptoms of multiple neuritis."

There is rarely any difficulty in distinguishing the alcohol cases. The combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, is quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning, or he has diabetes.

Treatment.—Rest in bed is essential. In the acute cases with fever, the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the physician must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit-drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sores an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often, with the most extreme de-

formity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

II. NEUROMATA.

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances, as in the case which I reported, the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The true fascicular neuroma occurs in the form of the small subcutaneous painful tumor—*tubercula dolorosa*—which is situated on the nerves of the skin about the joints, sometimes on the face or on the breast. It is not always made up of nerve fibres, but may be, as shown by Hoggan, an adenomatous growth of the sweat glands.

The true neuromata, as a rule, are not painful, and occasionally are found associated with the nerve fibres in various regions. Those which develop at the ends and along the course of the nerves of the stump after amputation consist of connective tissue and of medullated and non-medullated nerve fibres. The most remarkable form is the *plexiform neuroma*, in which the various nerve cords are occupied by many hundreds of tumors. The cases are usually congenital. The tumors occur in all the nerves of the body. One of the most remarkable is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. R. W. Smith's splendid monograph on neuromata has been reprinted this year (1898) by the New Sydenham Society.

Neuromata rarely cause symptoms, except the subcutaneous painful tumor or those in the amputation stump. Here they may be very painful and cause great distress. Motor symptoms are sometimes present, particularly a constant twitching. Epilepsy has sometimes been associated, and relief has followed removal of the growths.

The only available treatment is excision. The subcutaneous painful tumor does not return, and excision completely relieves the symptoms. On the other hand, the amputation neuromata may recur.

III. DISEASES OF THE CEREBRAL NERVES.

Olfactory Nerves and Tracts.

The functions of these nerves may be disturbed at their origin, in the nasal mucous membrane, at the bulb, in the course of the tract, or at the centres in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

(*a*) *Subjective Sensations; Parosmia.*—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampi. In rare instances, after injury of the head the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

(*b*) *Increased sensitiveness*, or *hyperosmia*, occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

(*c*) *Anosmia; Loss of the Sense of Smell.*—This may be produced by: (1) Affections of the origin of the nerves in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(2) The lesions of the bulb or of the tracts. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the olfactory tracts may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, possibly owing to atrophy of the nerves.

(3) Lesions of the olfactory centres. There are congenital cases in which the structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which anosmia has been associated with disease in the hemisphere. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus. Flechsig describes (1) a frontal centre in the base of the frontal lobe and (2) a temporal centre in the uncus.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in

brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopical examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

Optic Nerve and Tract.

(1) *Lesions of the Retina.*

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye-grounds. Only a brief reference can here be made to the more important of the appearances.

(*a*) **Retinitis.**—This occurs in certain general affections, more particularly in Bright's disease, syphilis, leukæmia, and anæmia. The common feature in all these states is the occurrence of hæmorrhage and the development of opacities. There may also be a diffuse cloudiness due to effusion of serum. The hæmorrhages are in the layer of nerve fibres. They vary greatly in size and form, but often follow the course of vessels. When recent the color is bright red, but they gradually change and old hæmorrhages are almost black. The white spots are due either to fibrinous exudate or to fatty degeneration of the retinal elements, and occasionally to accumulation of leucocytes or to a localized sclerosis of the retinal elements. The more important of the forms of retinitis to be recognized are:

Albuminuric retinitis, which occurs in chronic nephritis, particularly in the interstitial or contracted form. The percentage of cases affected is from 15 to 25. There are instances in which these retinal changes are associated with the granular kidney at a stage when the amount of albumen may be slight or transient; but in all such instances it will be found that there is a marked arterio-sclerosis. Gowers recognizes a degenerative form (most common), in which, with the retinal changes, there may be scarcely any alteration in the disk; a hæmorrhagic form, with many hæmorrhages and but slight signs of inflammation; and an inflammatory form, in which there is much swelling of the retina and obscuration of the disk. It is noteworthy that in some instances the inflammation of the optic nerve predominates over the retinal changes, and one may be in doubt for a time whether the condition is really associated with the renal changes or dependent upon intracranial disease.

Syphilitic Retinitis.—In the acquired form this is less common than choroiditis. In inherited syphilis *retinitis pigmentosa* is sometimes met with.

Retinitis in Anæmia.—It has long been known that a patient may become blind after a large hæmorrhage, either suddenly or within two or three days, and in one or both eyes. Occasionally the loss may be permanent and complete. In some of these instances a neuro-retinitis has been found, probably sufficient to account for the symptoms. In the more chronic anæmias, particularly in the pernicious form, retinitis is common, as determined first by Quincke.

In *malaria* retinitis or neuro-retinitis may be present, as noted by

Stephen Mackenzie. It is seen only in the chronic cases with anæmia, and in my experience is not nearly so common proportionately as in pernicious anæmia.

Leukæmic Retinitis.—In this affection the retinal veins are large and distended; there is also a peculiar retinitis, as described by Liebreich. It is not very common. It existed in only 3 of 10 cases of which I have notes of examination of the retina. There are numerous hæmorrhages and white or yellow areas, which may be large and prominent. In one of my cases the retina post mortem was dotted with many small, opaque, white spots, looking like little tumors, the larger of which had a diameter of nearly 2 mm. In Case 13 of my series the leukæmia was diagnosed by Norris and De Schweinitz, at whose clinic the patient had applied on account of failing vision, from the condition of the eye-grounds alone.

Retinitis is also found occasionally in diabetes, in purpura, in chronic lead poisoning, and sometimes as an idiopathic affection.

(*b*) **Functional Disturbances of Vision.**—(1) *Toxic Amaurosis.*—This occurs in uræmia and may follow convulsions or come on independently. The condition, as a rule, persists only for a day or two. This form of amaurosis occurs in poisoning by lead, alcohol, and occasionally by quinine. It seems more probable that the poisons act on the centres and not on the retina.

(2) *Tobacco Amblyopia.*—The loss of sight is usually gradual, equal in both eyes, and affects particularly the centre of the field of vision. The eye-grounds may be normal, but occasionally there is congestion of the disks. On testing the color fields a central scotoma for red and green is found in all cases. Ultimately, if the use of tobacco is continued, organic changes may develop with atrophy of the disk.

(3) *Hysterical Amaurosis.*—More frequently this is loss of acuteness of vision—amblyopia—but the loss of sight in one or both eyes may apparently be complete. The condition will be mentioned subsequently under hysteria.

(4) *Night-blindness—nyctalopia*—the condition in which objects are clearly seen during the day or by strong artificial light, but become invisible in the shade or in twilight, and *hemeralopia*, in which objects cannot be clearly seen without distress in daylight or in a strong artificial light, but are readily seen in a deep shade or in twilight, are functional anomalies of vision which rarely come under the notice of the physician. It may occur in epidemic form.

(5) *Retinal hyperæsthesia* is sometimes seen in hysterical women, but is not found frequently in actual retinitis. I have seen it once, however, in albuminuric retinitis, and once, in a marked degree, in a patient with aortic insufficiency, in whose retinæ there were no signs other than the throbbing arteries.

(2) *Lesions of the Optic Nerve.*

(*a*) **Optic Neuritis** (*Papillitis; Choked Disk*).—In the first stage there is congestion of the disk and the edges are blurred and striated. In the second stage, the congestion is more marked, the swelling increases, the

Nyctalopia

All communications to this Office should be addressed "To the Surgeon General, U. S. Army, Washington, D. C."

Subject:

WAR DEPARTMENT,
SURGEON GENERAL'S OFFICE,
U. S. ARMY MEDICAL MUSEUM AND LIBRARY,
Corner 7th and B Streets S. W.,

Washington, D. C., December 18, 1900., *189* .

Dear Doctor Osler:

I do not know whether you are collecting material for a notice of Greenhill, but meeting with a characteristic paper of his, I subjoin the reference. It is on the much befogged question of "nyctalopia" and "hemeralopia" and their supposed mugwumpery. These journal references are easily overlooked.

Very truly yours,

Richard Derby

These journal references are easily overlooked.

Very truly yours.

Robert Fletcher,

On the meaning of the words "nyctalopia" and "hemeralopia:" with a critical examination of the use of these words in the ancient Greek and Latin authors.
Ophth. Hosp. Rep.,Lond., 1880-82, x, 284-292.

striation also is more visible. The physiological cupping disappears and hæmorrhages are not uncommon. The arteries present little change, the veins are dilated, and the disk may swell greatly. In slight grades of inflammation the swelling gradually subsides and occasionally the nerve recovers completely. In instances in which the swelling and exudate are very great, the subsidence is slow, and when it finally disappears there is complete atrophy of the nerve. The retina not infrequently participates in the inflammation, which is then a neuro-retinitis.

This condition is of the greatest importance in diagnosis. It may exist in its early stages without any disturbance of vision, and even with extensive papillitis the sight may for a time be good.

Optic neuritis is seen occasionally in anæmia and lead poisoning, more commonly in Bright's disease as neuro-retinitis. It occurs occasionally as a primary idiopathic affection. The frequent connection with intracranial disease, particularly tumor, makes its presence of great value to practitioners. The nature of the growth is without influence. In over 90 per cent of such instances the papillitis is bilateral. It is also found in meningitis, either the tuberculous or the simple form. In meningitis it is easy to see how the inflammation may extend down the nerve sheath. In the case of tumor it was thought at first that a choked disk resulted from increased pressure within the skull. It is now more commonly regarded, however, as a descending neuritis.

(*b*) **Optic Atrophy.**—This may be: (1) A primary affection. There is an hereditary form, in which the disease has developed in all the males of a family shortly after puberty. A large number of the cases of primary atrophy are associated with spinal disease, particularly locomotor ataxia. Other causes which have been assigned for the primary atrophy are cold, sexual excesses, diabetes, the specific fevers, alcohol, and lead.

(2) Secondary atrophy results from cerebral diseases, pressure on the chiasma or on the nerves, or, most commonly of all, as a sequence of papillitis.

The ophthalmoscopic appearances are different in the cases of primary and secondary atrophy. In the former, the disk has a gray tint, the edges are well defined, and the arteries look almost normal; whereas in the consecutive atrophy the disk has a staring opaque-white aspect, with irregular outlines, and the arteries are very small.

The symptom of optic atrophy is loss of sight, proportionate to the damage in the nerve. The change is in three directions: "(1) Diminished acuity of vision; (2) alteration in the field of vision; and (3) altered perception of color" (Gowers). The outlook in primary atrophy is bad.

(3) *Affections of the Chiasma and Tract.*

At the chiasma the optic nerves undergo partial decussation. Each optic tract, as it leaves the chiasma, contains nerve fibres which originate in the retinæ of both eyes. Thus, of the fibres of the right tract, part have come through the chiasma without decussating from the temporal half of the right retina, the other and larger portion of the fibres of the tract

have decussated in the chiasma, coming as they do from the left optic nerve and the nasal half of the retina on the left side. The fibres which cross are in the middle portion of the chiasma, while the direct fibres are on each side. The following are the most important changes which ensue in lesions of the tract and of the chiasma:

(*a*) *Unilateral Affection of Tract.*—If on the right side, this produces loss of function in the temporal half of the retina on the right side, and on the nasal half of the retina on the left side, so that there is only half vision, and the patient is blind to objects on the left side. This is termed homonymous hemianopia or lateral hemianopia. The fibres passing to the right half of each retina being involved, necessarily the left half of each visual field is blind. The hemianopia may be partial and only a portion of the half field may be lost. The unaffected visual fields may have the normal extent, but in some instances there is considerable reduction. When the left half of one field and the right half of the other, or *vice versa*, are blind, the condition is known as heteronymous hemianopia.

(*b*) *Disease of the Chiasma.*—(1) A lesion involves, as a rule, chiefly the central portion, in which the decussating fibres pass which supply the inner or nasal halves of the retinæ, producing in consequence loss of vision in the outer half of each field, or what is known as temporal hemianopia.

(2) If the lesion is more extensive it may involve not only the central portion, but also the direct fibres on one side of the commissure, in which case there would be total blindness in one eye and temporal hemianopia in the other.

(3) Still more extensive disease is not infrequent from pressure of tumors in this region, the whole chiasma is involved, and total blindness results. The different stages in the process may often be traced in a single case from temporal hemianopia, then complete blindness in one eye with temporal hemianopia in the other, and finally complete blindness.

(4) A limited lesion of the outer part of the chiasma involves only the direct fibres passing to the temporal halves of the retinæ and inducing blindness in the nasal field, or, as it is called, nasal hemianopia. This, of course, is extremely rare. Double nasal hemianopia may occur as a manifestation of tabes and in tumors involving the outer fibres of each tract.

(4) *Affections of the Tract and Centres.*

The optic tract crosses the crus (cerebral peduncle) to the hinder part of the optic thalamus and divides into two portions, one of which (the lateral root) goes to the pulvinar of the thalamus, the lateral geniculate body, and to the anterior quadrigeminal body (superior colliculus). From these parts, in which the lateral root terminates, fibres pass into the posterior part of the internal capsule and enter the occipital lobe, forming the fibres of the optic radiation, which terminate in and about the cuneus, the region of the visual perceptive centre. The fibres of the medial division of the tract pass to the medial geniculate body and to the posterior quadrigeminal body. The medial root contains the fibres of the commissura inferior of v. Gudden, which are believed to have no connection with the

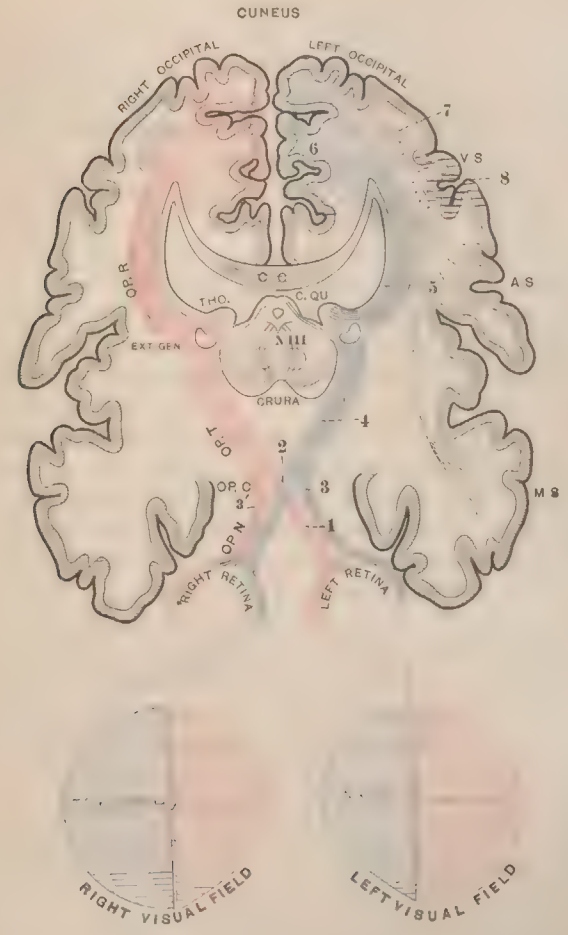

FIG. 11.—Diagram of visual paths. (From Vialet, modified.) OP. N., Optic nerve. OP. C., Optic chiasm. OP. T., Optic tract. OP. R., Optic radiations. GEN., Geniculate body. THO., Optic thalamus. C. QU., Corpora quadrigemina. C. C., Corpus callosum. V. S., Visual speech centre. A. S., Auditory speech centre. M. S., Motor speech centre. A lesion at 1 causes blindness of that eye; at 2, bi-temporal hemianopia; at 3, nasal hemianopia. Symmetrical lesions at 3 and 3′ would cause bi-nasal hemianopia; at 4, hemianopia of both eyes, with hemianopic pupillary inaction; at 5 and 6, hemianopia of both eyes, pupillary reflexes normal; at 7, amblyopia, especially of opposite eye; at 8, on left side, word-blindness.

retinæ. It is still held by some physiologists that the cortical visual centre is not confined to the occipital lobe alone, but embraces the occipito-angular region.

A lesion of the fibres of the optic path anywhere between the cortical centre and the chiasma will produce hemianopia. The lesion may be situated: (*a*) In the optic tract itself. (*b*) In the region of the thalamus, lateral geniculate body, and the corpora quadrigemina, into which the larger part of each tract enters. (*c*) A lesion of the fibres passing from the centres just mentioned to the occipital lobe. This may be either in the hinder part of the internal capsule or the white fibres of the optic radiation. (*d*) Lesion of the cuneus. Bilateral disease of the cuneus may result in total blindness. (*e*) There is clinical evidence to show that lesion of the angular gyrus may be associated with visual defect, not so often hemianopia as crossed amblyopia, dimness of vision in the opposite eye, and great contraction in the field of vision. Lesions in this region are associated with mind blindness, a condition in which there is failure to recognize the nature of objects.

The effects of lesions in the optic nerve in different situations from the retinal expansion to the brain cortex are as follows: (1) Of the optic nerve —total blindness of the corresponding eye; (2) of the optic chiasma, either temporal hemianopia, if the central part alone is involved, or nasal hemianopia, if the lateral region of each chiasma is involved; (3) lesion of the optic tract between the chiasma and the lateral geniculate body, produces lateral hemianopia; (4) lesion of the central fibres of the nerve between the geniculate bodies and the cerebral cortex produces lateral hemianopia; (5) lesion of the cuneus causes lateral hemianopia; and (6) lesion of the angular gyrus may be associated with hemianopia, sometimes crossed amblyopia, and the condition known as mind blindness. (See Fig. 11, with accompanying explanation.)

Diagnosis.—The student or practitioner must have a clear idea of the physiology of the nerve centres before he can appreciate the symptoms or undertake the diagnosis of lesions of the optic nerve. Having determined the presence of hemianopia, the question arises as to the situation of the lesion, whether in the tract between the chiasma and the geniculate bodies or in the central portion of the fibres between these bodies and the visual centres. This can be determined in some cases by the test known as Wernicke's *hemiopic pupillary inaction*. The pupil reflex depends on the integrity of the retina or receiving membrane, on the fibres of the optic nerve and tract which transmit the impulse, and the nerve centre at the termination of the optic tract which receives the impression and transmits it to the third nerve along which the motor impulses pass to the iris. If a bright light is thrown into the eye and the pupil reacts, the integrity of this reflex arc is demonstrated. It is possible in cases of lateral hemianopia so to throw the light into the eye that it falls upon the blind half of the retina. If when this is done the pupil contracts, the indication is that the reflex arc above referred to is perfect, by which we mean that the optic nerve fibres from the retinal expansion to the centre, the centre itself, and the third nerve are uninvolved. In such a case the conclusion

would be justified that the cause of the hemianopia was central; that is, situated beyond the geniculate body, either in the fibres of the optic radiation or in the visual cortical centres. If, on the other hand, when the light is carefully thrown on the hemiopic half of the retina, the pupil remains inactive, the conclusion is justifiable that there is interruption in the path between the retina and the nucleus of the third nerve, and that the hemianopia is not central, but dependent upon a lesion situated in the optic tract. This test of Wernicke's is sometimes difficult to obtain. It is best performed as follows: "The patient being in a dark or nearly dark room with the lamp or gas-light behind his head in the usual position, I bid him look over to the other side of the room, so as to exclude accommodative iris movements (which are not necessarily associated with the reflex). Then I throw a faint light from a plane mirror or from a large concave mirror, held well out of focus, upon the eye and note the size of the pupil. With my other hand I now throw a beam of light, focussed from the lamp by an ophthalmoscopic mirror, directly into the optical centre of the eye; then laterally in various positions, and also from above and below the equator of the eye, noting the reaction at all angles of incidence of the ray of light." (Seguin.)

The significance of hemianopia varies. There is a functional hemianopia associated with migraine and hysteria. In a considerable proportion of all cases there are signs of organic brain-disease. In a certain number of instances of slight lesions of the occipital lobe hemiachromatopsia has been observed. The homonymous halves of the retina as far as the fixation point are dulled, or blind for colors. Hemiplegia is common, in which event the loss of power and blindness are on the same side. Thus, a lesion in the left hemisphere involving the motor tract produces right hemiplegia, and when the fibres of the optic radiation are involved in the internal capsule, there is also lateral hemianopia, so that objects in the field of vision to the right are not perceived. Hemianæsthesia is not uncommon in such cases, owing to the close association of the sensory and visual tracts at the posterior part of the internal capsule. Certain forms of aphasia also occur in many of the cases.

The optic aphasia of Freund may be mentioned here. The patient after an apoplectic attack, though able to recognize ordinary objects shown to him is unable to name them correctly. If he be permitted to touch the object he may be able to name it quickly and correctly. Freund's optic aphasia differs from mind-blindness, since in the latter affection the objects seen are not recognized. Optic aphasia, like word-blindness, never occurs alone, but is always associated with hemianopia, or mind-blindness, and often also with word-deafness. In the cases which have thus far come to autopsy there has always been a lesion in the white matter of the occipital lobe on the left side.

Motor Nerves of the Eyeball.

Third Nerve (*Nervus oculomotorius*).—The nucleus of origin of this nerve is situated in the floor of the aqueduct of Sylvius; the nerve passes

through the crus at the side of which it emerges. Passing along the wall of the cavernous sinus, it enters the orbit through the sphenoidal fissure and supplies, by its superior branch, the levator palpebræ superioris and the superior rectus, and by its inferior branch the internal and inferior recti muscles and the inferior oblique. Branches pass to the ciliary muscle and the constrictor of the iris. Lesions may affect the nucleus or the nerve in its course and cause either paralysis or spasm.

Paralysis.—A nuclear lesion is usually associated with the disease of the centres for the other eye muscles, producing a condition of general ophthalmoplegia. More commonly the nerve itself is involved in its course, either by meningitis, gummata, or aneurism, or is attacked by a neuritis, as in diphtheria and locomotor ataxia. Complete paralysis of the third nerve is accompanied by the following symptoms:

Paralysis of all the muscles, except the superior oblique and external rectus, by which the eye can be moved outward and a little downward and inward. There is divergent strabismus. There is ptosis or drooping of the upper eyelid, owing to paralysis of the levator palpebræ. The pupil is usually dilated. It does not contract to light, and the power of accommodation is lost. The most striking features of this paralysis are the external strabismus, with diplopia or double vision, and the ptosis. In very many cases the affection of the third nerve is partial. Thus the levator palpebræ and the superior rectus may be involved together, or the ciliary muscles and the iris may be affected and the external muscles may escape.

There is a remarkable form of recurring oculo-motor paralysis affecting chiefly women, and involving all the branches of the nerve. In some cases the attacks have come on at intervals of a month; in others a much longer period has elapsed. The attacks may persist throughout life. They are sometimes associated with pain in the head and sometimes with migraine. Mary Sherwood has collected from the literature 23 cases.

Ptosis is a common and important symptom in nervous affections. We may here briefly refer to the conditions under which it may occur: (*a*) A congenital, incurable form, which is frequently seen; (*b*) the form associated with definite lesion of the third nerve, either in its course or at its nucleus. This may come on with paralysis of the superior rectus alone or with paralysis of the internal and inferior recti as well. (*c*) There are instances of complete or partial ptosis associated with cerebral lesions without any other branch of the third nerve being paralyzed. The exact position of the cortical centre or centres is as yet unknown. (*d*) Hysterical ptosis, which is double and occurs with other hysterical symptoms. (*e*) Pseudo-ptosis, due to affection of the sympathetic nerve, is associated with symptoms of vaso-motor palsy, such as elevation of the temperature on the affected side with redness and œdema of the skin. Contraction of the pupil exists on the same side and the eyeball appears rather to have shrunk into the orbit. (*f*) In idiopathic muscular atrophy, when the face muscles are involved, there may be marked bilateral ptosis. And, lastly, in weak, delicate women there is often to be seen a transient ptosis, particularly in the morning.

Among the most important of the symptoms of the third-nerve paralysis are those which relate to the ciliary muscle and iris.

Cycloplegia, paralysis of the ciliary muscle, causes loss of the power of accommodation. Distant vision is clear, but near objects cannot be properly seen. In consequence the vision is indistinct, but can be restored by the use of convex glasses. This may occur in one or in both eyes; in the latter case it is usually associated with disease in the nuclei of the nerve. Cycloplegia is an early and frequent symptom in diphtheritic paralysis and occurs also in tabes.

Iridoplegia, or paralysis of the iris, occurs in three forms (Gowers).

(*a*) *Accommodative iridoplegia*, in which the pupil does not diminish in size during the act of accommodation. To test for this the patient should look first at a distant and then at a near object in the same line of vision.

(*b*) *Reflex Iridoplegia.*—The path for the iris reflex is along the optic nerve and tract to its termination, then to the nucleus of the third nerve, and along the trunk of this nerve to the ciliary ganglion, and so through the ciliary nerves to the eyes. Each eye should be tested separately, the other one being covered. The patient should look at a distant object in a dark part of the room; then a light is brought suddenly in front of the eye at a distance of three or four feet, so as to avoid the effect of accommodation. Loss of this iris reflex with retention of the accommodation contraction is known as the Argyll Robertson pupil.

(*c*) *Loss of the Skin Reflex.*—If the skin of the neck is pinched or pricked the pupil dilates reflexly, the afferent impulses being conveyed along the cervical sympathetic. Erb pointed out that this skin reflex is lost usually in association with the reflex contraction, but the two are not necessarily conjoined. In iridoplegia the pupils are often small, particularly in spinal disease, as in the characteristic small pupils of tabes—spinal myosis. Iridoplegia may coexist with a pupil of medium size.

Inequality of the pupils—aniscoria—is not infrequent in progressive paresis and in tabes. It may also occur in perfectly healthy individuals.

Spasm.—Occasionally in meningitis and in hysteria there is spasm of the muscles supplied by the third nerve, particularly the internal rectus and the levator palpebræ. The clonic rhythmical spasm of the eye muscles is known as *nystagmus*, in which there is usually a bilateral, rhythmical, involuntary movement of the eyeballs. The condition is met with in many congenital and acquired brain lesions, in albinism, and sometimes in coal-miners.

Fourth Nerve (*Nervus trochlearis*).—This supplies the superior oblique muscle. In its course around the outer surface of the crus and in its passage into the orbit it is liable to be compressed by tumors, by aneurism, or in the exudation of basilar meningitis. Its nucleus in the upper part of the fourth ventricle may be involved by tumors or undergo degeneration with the other ocular nuclei. The superior oblique muscle acts in such a way as to direct the eyeball downward and rotates it slightly. The paralysis causes defective downward and inward movement, often too slight to be

noticed. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down.

Sixth Nerve (*Nervus abducens*).—This nerve emerges at the junction of the pons and medulla, then, passing forward, it enters the orbit and supplies the external rectus muscle. It is affected by meningitis at the base, by gummata or other tumors, and sometimes by cold. There is internal strabismus, and the eye cannot be turned outward. Diplopia occurs on looking toward the paralyzed side.

"When the nucleus is affected there is, in addition to paralysis of the external rectus, inability of the internal rectus of the opposite eye to turn that eye inward. As a consequence of this the axes of the eyes are kept parallel and both are conjugately deviated to the opposite side, away from the side of lesion. The reason of this is that the nucleus of the sixth nerve sends fibres up in the pons to that part of the nucleus of the opposite third nerve which supplies the internal rectus. We thus have paralysis of the internal rectus without the nucleus of the third nerve being involved, owing to its receiving its nervous impulses for parallel movement from the sixth nucleus of the opposite side. As the sixth nucleus is in such proximity to the facial nerve in the substance of the pons, it is frequently found that the whole of the face on the same side is paralyzed, and gives the electrical reaction of degeneration, so that with a lesion of the *left* sixth nucleus there is conjugate deviation of both eyes to the *right*—i. e., paralysis of the left external and the right internal rectus, and sometimes complete paralysis of the *left* side of the face" (Beevor).

General Features of Paralysis of the Motor Nerves of the Eye.—Gowers divides them into five groups:

(*a*) *Limitation of Movement.*—Thus, in paralysis of the external rectus, the eyeball cannot be moved outward. When the paralysis is incomplete the movement is deficient in proportion to the degree of the palsy.

(*b*) *Strabismus.*—The axes of the eyes do not correspond. Thus, paralysis of the internal rectus causes a divergent squint; of the external rectus, a convergent squint. At first this is only evident when the eyes are moved in the direction of the action of the weak muscle, but may become constant by the contraction of the opposing muscle. The deviation of the axis of the affected eye from parallelism with the other is called the primary deviation.

(*c*) *Secondary Deviation.*—If, while the patient is looking at an object, the sound eye is covered, so that he fixes the object looked at with the affected eye only, the sound eye is moved still further in the same direction—e. g., outward—with paralysis of the opposite internal rectus. This is known as secondary deviation. It depends upon the fact that, if two muscles are acting together, when one is weak and an effort is made to contract it, the increased effort—innervation—acts powerfully upon the other muscle, causing an increased contraction.

(*d*) *Erroneous Projection.*—"We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball

as indicated to us by the innervation we give to the ocular muscles" (Gowers). With the eyes at rest in the mid-position, an object at which we are looking is directly opposite our face. Turning the eyes to one side, we recognize that object in the middle of the field or to the side of this former position. We estimate the degree by the amount of movement of the eyes, and when the object moves and we follow it we judge of its position by the amount of movement of the eyeballs. When one ocular muscle is weak, the increased innervation gives the impression of a greater movement of the eye than has really taken place. The mind, at the same time, receives the idea that the object is further on one side than it really is, and in an attempt to touch it the finger may go beyond it. As the equilibrium of the body is in a large part maintained by a knowledge of the relation of external objects to it obtained by the action of the eye muscles, this erroneous projection resulting from paralysis disturbs the harmony of these visual impressions and may lead to giddiness—ocular vertigo.

(*e*) *Double Vision.*—This is one of the most disturbing features of paralysis of the eye muscles. The visual axes do not correspond, so that there is a double image—diplopia. That seen by the sound eye is termed the true image; that by the paralyzed eye, the false. In simple or homonymous diplopia the false image is "on the same side of the other as the eye by which it is seen." In crossed diplopia it is on the other side. In convergent squint the diplopia is simple; in divergent it is crossed.

Ophthalmoplegia.—Under this term is described a chronic progressive paralysis of the ocular muscles. Two forms are recognized—ophthalmoplegia *externa* and ophthalmoplegia *interna*. The conditions may occur separately or together and are described by Gowers under nuclear ocular palsy.

Ophthalmoplegia externa.—The condition is one of more or less complete palsy of the external muscles of the eyeball, due usually to a slow degeneration in the nuclei of the nerves, but sometimes to pressure of tumors or to basilar meningitis. It is often, but not necessarily, associated with ophthalmoplegia interna. Siemerling, in a monograph on the subject, states that 62 cases are on record. In only 11 of these could syphilis be positively determined. The levator muscles of the eyelids and the superior recti are first involved, and gradually the other muscles, so that the eyeballs are fixed and the eyelids droop. There is sometimes slight protrusion of the eyeballs. The disease is essentially chronic and may last for many years. It is found particularly in association with general paralysis, locomotor ataxia, and in progressive muscular atrophy. Mental disorders were present in 11 of the 62 cases. With it may be associated atrophy of the optic nerve and affections of other cerebral nerves. Occasionally, as noted by Bristowe, it may be functional.

Ophthalmoplegia interna.—Jonathan Hutchinson applied this term to a progressive paralysis of the internal ocular muscles, causing loss of pupillary action and the power of accommodation. When the internal and external muscles are involved the affection is known as total ophthalmoplegia, and in a majority of the cases the two conditions are associated. In some instances the internal form may depend upon disease of the ciliary ganglion.

While, as a rule, ophthalmoplegia is a chronic process, there is an acute form associated with hæmorrhagic softening of the nuclei of the ocular muscles. There is usually marked cerebral disturbance. It was to this form that Wernicke gave the name polio-encephalitis superior.

Treatment of Ocular Palsies.—It is important to ascertain, if possible, the cause. The forms associated with locomotor ataxia are obstinate, and resist treatment. Occasionally, however, a palsy, complete or partial, may pass away spontaneously. The group of cases associated with chronic degenerative changes, as in progressive paresis and bulbar paralysis, is little affected by treatment. On the other hand, in syphilitic cases, mercury and iodide of potassium are indicated, and are often beneficial. Arsenic and strychnia, the latter hypodermically, may be employed. In any case in which the onset is acute, with pain, hot fomentations and counter-irritation or leeches applied to the temple give relief. The direct treatment by electricity has been extensively employed, but probably without any special effect. The diplopia may be relieved by the use of prisms, or it may be necessary to cover the affected eye with an opaque glass.

Fifth Nerve (*Nervus trigeminus*).

Paralysis may result from: (*a*) Disease of the pons, particularly hæmorrhage or patches of sclerosis. (*b*) Injury or disease at the base of the brain. Fracture rarely involves the nerve; on the other hand, meningitis, acute or chronic, and caries of the bone are not uncommon causes. (*c*) The branches may be affected as they pass out—the first division by tumors pressing on the cavernous sinus or by aneurism; the second and third divisions by growths which invade the spheno-maxillary fossa. (*d*) Primary neuritis, which is rare.

Symptoms.—(*a*) *Sensory Portion.*—Disease of the fifth nerve may cause loss of sensation in the parts supplied, including the half of the face, the corresponding side of the head, the conjunctiva, the mucosa of the lips, tongue, hard and soft palate, and of the nose of the same side. The anæsthesia may be preceded by tingling or pain. The muscles of the face are also insensible and the movements may be slower. The sense of smell is interfered with. There is disturbance of the sense of taste. There are, in addition, *trophic* changes; the salivary, lachrymal, and buccal secretions may be lessened, abrasions of the mucous membranes heal slowly, and the teeth may become loose. The eye inflames, the corneæ become cloudy and may ulcerate. It was formerly held that these symptoms only occurred when the Gasserian ganglion was affected, but of late years this has been completely removed for obstinate neuralgia without producing any trophic disturbance. This apparent contradiction is not yet explained. Herpes may develop in the region supplied by the nerve, usually the upper branch, and is associated with much pain, which may be peculiarly enduring, lasting for months or years (Gowers). In herpes zoster with the neuritis there may be slight enlargement of the cervical glands.

(*b*) *Motor Portion.*—The inability to use the muscles of mastication on the affected side is the distinguishing feature of paralysis of this portion of

S. Browning has called attention to the special liability to facial paralysis in certain families. (Albany Med. Annals June - 1899).

the nerve. It is recognized by placing the finger on the masseter and temporal muscles, and, when the patient closes the jaw, the feebleness of their contraction is noted. If paralyzed, the external pterygoid cannot move the jaw toward the unaffected side; and when depressed, the jaw deviates to the paralyzed side. The motor paralysis of the fifth nerve is almost invariably a result of involvement of the nerve after it has left the nucleus. Cases, however, have been associated with cortical lesions. Hirt concludes, from his case, that the cortical motor centre for the trigeminus is in the neighborhood of the lower third of the anterior central convolution.

Spasm of the Muscles of Mastication.—Trismus, the masticatory spasm of Romberg, may be tonic or clonic, and is either an associated phenomenon in general convulsions or, more rarely, an independent affection. In the tonic form the jaws are kept close together—lock-jaw—or can be separated only for a short space. The muscles of mastication can be seen in contraction and felt to be hard; the spasm is often painful. This tonic contraction is an early symptom in tetanus, and is sometimes seen in tetany. A form of this tonic spasm occurs in hysteria. Occasionally trismus follows exposure to cold, and is said to be due to reflex irritation from the teeth, the mouth, or caries of the jaw. It may also be a symptom of organic disease due to irritation near the motor nucleus of the fifth nerve.

Clonic spasm of the muscles supplied by the fifth occurs in the form of rapidly repeated contractions, as in "chattering teeth." This is rare apart from general conditions, though cases are on record, usually in women late in life, in whom this isolated clonic spasm of the muscles of the jaw has been found. In another form of clonic spasm sometimes seen in chorea, there are forcible single contractions. Gowers mentions an instance of its occurrence as an isolated affection.

(*c*) *Gustatory.*—Loss of the sense of taste in the anterior two thirds of the tongue, as a rule, follows paralysis of the fifth nerve. The gustatory fibres pass from the chorda tympani to the lingual branch of the fifth. Disease of the fifth nerve is, however, not always associated with loss of taste in the anterior part of the tongue, in which case either the taste fibres escape, or the disease is within the pons where these fibres are separate from those of sensation. It may be that the nervus intermedius of Wrisberg carries the taste fibres.

The *diagnosis* of disease of the trifacial nerve is rarely difficult. It must be remembered that the preliminary pain and hyperæsthesia are sometimes mistaken for ordinary neuralgia. The loss of sensation and the palsy of the muscles of mastication are readily determined.

Treatment.—When the pain is severe morphia may be required and local applications are useful. If there is a suspicion of syphilis, appropriate treatment should be given. Faradization is sometimes beneficial.

Facial Nerve.

Paralysis (*Bell's Palsy*).—The facial or seventh may be paralyzed by (1) lesions of the cortex—supranuclear palsy; (2) lesions of the nucleus

itself; or (3) involvement of the nerve trunk in its tortuous course within the pons and through the wall of the skull.

1. *Supranuclear paralysis,* due to lesion of the cortex or of the facial fibres in the corona radiata or internal capsule, is, as a rule, associated with hemiplegia. It may be caused by tumors, abscess, chronic inflammation, or softening in the cortex or in the region of the internal capsule. It is distinguished from the peripheral form by well-marked characters—the persistence of the normal electrical excitability of both nerves and muscles and the absence of involvement of the upper branches of the nerve, so that the orbicularis palpebrarum and frontalis muscles are spared. In rare instances these muscles are paralyzed. A third difference is that in this form the voluntary movements are more impaired than the emotional. There are instances of cortical facial paralysis—monoplegia facialis—associated with lesions in the centre for the face muscles in the lower Rolandic region. Isolated paralysis, due to involvement of the nerve fibres in their path to the nucleus, is uncommon. In the great majority of cases supranuclear facial paralysis is part of a hemiplegia. Paralysis is on the same side as that of the arm and leg because the facial muscles bear precisely the same relation to the cortex as the spinal muscles. The nuclei of origin on either side of the middle line in the medulla are united by decussating fibres with the cortical centre on the opposite side (see Fig. 11). A few fibres reach the nucleus from the cerebral cortex of the same side (Mellus, Hoche).

2. The *nuclear paralysis* caused by lesions of the nerve centres in the medulla is not common alone; but is seen occasionally in tumors, chronic softening, and hæmorrhage. We have had one instance of its involvement in anterior polio-myelitis. In diphtheria this centre may also be involved. The symptoms are practically similar to those of an affection of the nerve fibre itself—infranuclear paralysis.

3. *Involvement of the Nerve Trunk.*—Paralysis may result from:

(*a*) Involvement of the nerve as it passes through the pons—that is, between its nucleus in the floor of the fourth ventricle and the point of emergence in the postero-lateral aspect of the pons. The specially interesting feature in connection with involvement of this part is the production of what is called alternating or *crossed paralysis,* the face being involved on the same side as the lesion, and the arm and leg on the opposite side, since the motor path is involved above the point of decussation in the medulla (Fig. 11). This occurs only when the lesion is in the lower section of the pons. A lesion in the upper half of the pons involves the fibres not of the outgoing nerve on the same side, but of the fibres from the hemispheres before they have crossed to the nucleus of the opposite side. In this case there would of course be, as in hemiplegia, paralysis of the face and limbs on the side opposite to the lesion. The palsy, too, would resemble the cerebral form, involving only the lower fibres of the facial nerve.

(*b*) The nerve may be involved at its point of emergence by tumors, gummata, meningitis, or occasionally may be injured in fracture of the base.

(*c*) In passing through the Fallopian canal the nerve may be involved in disease of the ear, particularly by caries of the bone in otitis media.

This is a common cause in children. I have seen two instances follow otitis in puerperal fever.

(*d*) As the nerve emerges from the styloid foramen it is exposed to injuries and blows which not infrequently cause paralysis. The fibres may be cut in the removal of tumors in this region, or the paralysis may be caused by pressure of the forceps in an instrumental delivery.

(*e*) Exposure to cold is the most common cause of facial paralysis, inducing a neuritis of the nerve within the Fallopian canal.

(*f*) Syphilis is not an infrequent cause, and the paralysis may develop early with the secondary symptoms.

(*g*) It may develop with herpes.

Facial diplegia is a rare condition occasionally found in affections at the base of the brain, lesions in the pons, simultaneous involvement of the nerves in ear disease, and in diphtheritic paralysis. Disease of the nuclei or symmetrical involvement of the cortex might also produce it. It may occur as a congenital affection. H. M. Thomas has described two cases in one family.

Symptoms.—In the peripheral facial paralysis all the branches of the nerve are involved. The face on the affected side is immobile and can neither be moved at will nor participate in any emotional movements. The skin is smooth and the wrinkles are effaced, a point particularly noticeable on the forehead of elderly persons. The eye cannot be closed, the lower lid droops, and the eye waters. On the affected side the angle of the mouth is lowered, and in drinking the lips are not kept in close apposition to the glass, so that the liquid is apt to run out. In smiling or laughing the contrast is most striking, as the affected side does not move, which gives a curious unequal appearance to the two sides of the face. The eye cannot be closed nor can the forehead be wrinkled. In long-standing cases, when the reaction of degeneration is present, if the patient tries to close the eyes while looking fixedly at an object the lids on the sound side close firmly, but on the paralyzed side there is only a narrowing of the palpebral orifice, and the eye is turned upward and outward by the inferior oblique. On asking the patient to show his upper teeth, the angle of the mouth is not raised. In all these movements the face is drawn to the sound side by the action of the muscles. Speaking may be slightly interfered with, owing to the imperfection in the formation of the labial sounds. Whistling cannot be performed. In chewing the food, owing to the paralysis of the buccinator, particles collect on the affected side. The paralysis of the nasal muscles is seen on asking the patient to sniff. Owing to the fact that the lips are drawn to the sound side, the tongue, when protruded, looks as if it were pushed to the paralyzed side; but on taking its position from the incisor teeth, it will be found to be in the middle line. The reflex movements are lost in this peripheral form. It is usually stated that the palate is paralyzed on the same side and that the uvula deviates. Both Gowers and Hughlings Jackson deny the existence of this involvement in the great majority of cases, and Horsley and Beevor have shown that these parts are innervated by the accessory nerve to the vagus.

When the nerve is involved within the canal between the genu and the

origin of the chorda tympani, the sense of taste may be lost in the anterior part of the tongue on the affected side, owing probably to injury to the nervus intermedius of Wrisberg. When the nerve is damaged outside the skull the sense of taste is unaffected. Hearing is often impaired in facial paralysis, most commonly by preceding ear-disease. The paralysis of the stapedius muscle may lead to increased sensitiveness to musical notes. Herpes is sometimes associated with facial paralysis. Pain is not common, but there may be neuralgia about the ear. The face on the affected side may be swollen.

The *electrical reactions*, which are those of a peripheral palsy, have considerable importance from a prognostic standpoint. Erb's rules are as follows: If there is no change, either faradic or galvanic, the prognosis is good and recovery takes place in from fourteen to twenty days. If the faradic and galvanic excitability of the nerve is only lessened and that of the muscle increased to the galvanic current and the contraction formula altered (the contraction sluggish AnC>CC), the outlook is relatively good and recovery will probably take place in from four to six weeks; occasionally in from eight to ten. When the reaction of degeneration is present—that is, if the faradic and galvanic excitability of the nerves and the faradic excitability of the muscles are lost and the galvanic excitability of the muscle is quantitatively increased and qualitatively changed, and if the mechanical excitability is altered—the prognosis is relatively unfavorable and the recovery may not occur for two, six, eight, or even fifteen months.

The *course* of facial paralysis is usually favorable. The onset in the form following cold is very rapid, developing perhaps within twenty-four hours, but rarely is the paralysis permanent. Recurring attacks have been described; Sinkler mentions five. On the other hand, in the paralysis from injury, as by a blow on the mastoid process, the condition may remain. When permanent, the muscles are entirely toneless. In some instances contracture develops as the voluntary power returns, and the natural folds and the wrinkles on the affected side may be deepened, so that on looking at the face one at first may have the impression that the affected side is the sound one. This is corrected at once on asking the patient to smile, when it is seen which side of the face has the most active movement. Aretæus noted the difficulty sometimes experienced in determining which side was affected until the patient spoke or laughed.

The *diagnosis* of facial paralysis is usually easy. The distinction between the peripheral and central form is based on facts already mentioned.

Treatment.—In the cases which result from cold and are probably due to neuritis within the bony canal, hot applications first should be made; subsequently the thermo-cautery may be used lightly at intervals of a day or two over the mastoid process, or small blisters applied. If the ear is diseased, free discharge for the secretion should be obtained. The continuous current may be employed to keep up the nutrition of the muscles. The positive pole should be placed behind the ear, the negative one along the zygomatic and other muscles. The application can be made daily for a quarter of an hour and the patient can readily be taught to make it himself before the looking-glass. Massage of the muscles of the face is also

useful. A course of iodide of potassium may be given even when there is no indication of syphilis.

In some of the traumatic cases the possibility of surgical interference may be considered. In a patient with chronic otitis media of twenty-three years' duration and secondary mastoid disease Bloodgood operated in May, 1896. Complete facial paralysis followed. Eight weeks later the facial nerve was exposed in its canal and found to be almost completely severed. The ends were brought together and the wound allowed to fill with blood-clot, which organized. Four months later the patient had improved, and one year and six months from the operation the power had returned to all the muscles except the occipito-frontalis and the depressor of the lower lip. The response to galvanic and faradic currents was normal.

Spasm.—The spasm may be limited to a few or involve all the muscles innervated by the facial nerve and may be unilateral or bilateral.

It is known also by the name of mimic spasm or of convulsive tic. Several different affections are usually considered under the name of facial or mimic spasm, but we shall here speak only of the simple spasm of the facial muscles, either primary or following paralysis, and shall not include the cases of habit spasm in children, or the *tic convulsif* of the French.

Gowers recognizes two classes—one in which there is an organic lesion, and an idiopathic form. It is thought to be due also to reflex causes, such as the irritation from carious teeth or the presence of intestinal worms. The disease usually occurs in adults, whereas the habit spasm and the *tic convulsif* of the French, often confounded with it, are most common in children. True mimic spasm occasionally comes on in childhood and persists. In the case of a school-mate, the affection was marked as early as the eleventh or twelfth year and still continues. When the result of organic disease, there has usually been a lesion of the centre in the cortex, as in the case reported by Berkeley, or pressure on the nerve at the base of the brain by aneurism or tumor.

Symptoms.—The spasm may involve only the muscles around the eye—blepharospasm—in which case there is constant, rapid, quick action of the orbicularis palpebrarum, which, in association with photophobia, may be tonic in character. More commonly the spasm affects the lateral facial muscles with those of the eye, and there is constant twitching of the side of the face with partial closure of the eye. The frontalis is rarely involved. In aggravated cases the depressors of the angle of the mouth, the levator menti, and the platysma myoides are affected. This spasm is confined to one side of the face in a majority of cases, though it may extend and become bilateral. It is increased by emotional causes and involuntary movements of the face. As a rule, it is painless, but there may be tender points over the course of the fifth nerve, particularly the supraorbital branch. Tonic spasm of the facial muscle may follow paralysis, and is said to result occasionally from cold.

The outlook in facial spasm is always dubious. A majority of the cases persist for years and are incurable.

Treatment.—Sources of irritation should be looked for and removed. When a painful spot is present over the fifth nerve, blistering or the application of the thermo-cautery may relieve it. Hypodermic injections of strychnia may be tried, but are of doubtful benefit. Weir Mitchell recommends the freezing of the cheek for a few minutes daily or every second day with the spray, and this, in some instances, is beneficial. Often the relief is transient; the cases return, and at every clinic may be seen half a dozen or more of such patients who have run the gamut of all measures without material improvement. Operative interference may be resorted to in severe cases, although not much can be expected of it.

Auditory Nerve.

The eighth, known also as *portio mollis* of the seventh pair, passes from the ear through the internal auditory meatus, and in reality consists of two separate nerves—the cochlear and vestibular roots. These two roots have entirely different functions, and may therefore be best considered separately. The cochlear nerve is the one connected with the organ of Corti, and is concerned in hearing. The vestibular nerve is connected with the vestibule and semicircular canals, and has to do with the maintenance of equilibrium.

The Cochlear Nerve.

The cortical centre for hearing is in the temporo-sphenoidal lobe. Primary disease of the auditory nerve in its centre or intracranial course is uncommon. More frequently the terminal branches are affected within the labyrinth.

(*a*) *Affection of the Cortical Centre.*—In the monkey, experiments indicate that the superior temporal gyrus represents the centre for hearing. In man the cases of disease indicate that it has the same situation, as destruction of this gyrus on the left side results in word-deafness, which may be defined as an inability to understand the meaning of words, though they may still be heard as sounds. The central auditory path extending to the cortical centre from the terminal nuclei of the cochlear nerve may be involved and produce deafness. This may result from involvement of the lateral lemniscus from the presence of a tumor in the corpora quadrigemina, especially if it involve the posterior quadrigeminal bodies from a lesion of the internal geniculate body, or it may be associated with a lesion of the internal capsule.

(*b*) *Lesions of the nerve at the base* of the brain may result from the pressure of tumors, meningitis (particularly the cerebro-spinal form), hæmorrhage, or traumatism. A primary degeneration of the nerve may occur in locomotor ataxia. Primary disease of the terminal nuclei of the cochlear nerve (nucleus nervi cochlearis dorsalis and nucleus nervi cochlearis ventralis) is rare. By far the most interesting form results from epidemic cerebro-spinal meningitis, in which the nerve is frequently involved, causing permanent deafness. In young children the condition results in deaf-mutism.

(*c*) In a majority of the cases associated with auditory-nerve symptoms the lesion is in the internal ear, either primary or the result of extension of disease of the middle ear. Two groups of symptoms may be produced—hyperæsthesia and irritation and diminished function or nervous deafness.

(1) *Hyperæsthesia and Irritation.*—This may be due to altered function of the centre as well as of the nerve ending. True hyperæsthesia—hyperacusis—is a condition in which sounds, sometimes even those inaudible to other persons, are heard with great intensity. It occurs in hysteria and occasionally in cerebral disease. As already mentioned, in paralysis of the stapedius low notes may be heard with intensity. In dysæsthesia, or dysacusis, ordinary sounds cause an unpleasant sensation, as commonly happens in connection with headache, when ordinary noises are badly borne.

Tinnitus aurium is a term employed to designate certain subjective sensations of ringing, roaring, ticking, and whirring noises in the ear. It is a very common and often a distressing symptom. It is associated with many forms of ear-disease and may result from pressure of wax on the drum. It is rare in organic disease of the central connections of the nerve. Sudden intense stimulation of the nerve may cause it. A form not uncommonly met with in medical practice is that in which the patient hears a continual *bruit* in the ear, and the noise has a systolic intensification, usually on one side. I have twice been consulted by physicians for this condition under the belief that they had an internal aneurism. A systolic murmur may be heard occasionally on auscultation. It occurs in conditions of anæmia and neurasthenia. Subjective noises in the ear may precede an epileptic seizure and are sometimes present in migraine. In whatever form tinnitus exists, though slight and often regarded as trivial, it occasions great annoyance and often mental distress, and has even driven patients to suicide.

The *diagnosis* is readily made; but it is often extremely difficult to determine upon what condition the tinnitus depends. The relief of constitutional states, such as anæmia, neurasthenia, or gout, may result in cure. A careful local examination of the ear should always be made. One of the most worrying forms is the constant clicking, sometimes audible many feet away from the patient, and due probably to clonic spasm of the muscles connected with the Eustachian tube or of the levator palati. The condition may persist for years unchanged, and then disappear suddenly. The pulsating forms of tinnitus, in which the sound is like that of a systolic *bruit*, are almost invariably subjective, and it is very rare to hear anything with the stethoscope. It is to be remembered that in children there is a systolic brain murmur, best heard over the ear, and in some instances appreciable in the adult.

(2) *Diminished Function or Nervous Deafness.*—In testing for nervous deafness, if the tuning-fork cannot be heard when placed near the meatus, but the vibrations are audible by placing the foot of the tuning-fork against the temporal bone, the conclusion may be drawn that the deafness is not due to involvement of the nerve. The vibrations are conveyed through the temporal bone to the cochlea and vestibule. The watch may be used for the same purpose, and if the meatus is closed and the watch is heard

better in contact with the mastoid process than when opposite the open meatus, the deafness is probably not nervous. Practically, disturbance of the function of the auditory nerve is not a very frequent symptom in brain-disease, but in all cases the function of the nerve should be carefully tested.

The Vestibular Nerve.

The most frequent symptoms met with in association with disease of the vestibular nerve and its central connections are vertigo, nystagmus, and loss of coördination of the muscles of the head, neck, and eyes.

Auditory Vertigo—Ménière's Disease.—In 1861 Ménière, a French physician, described an affection characterized by noises in the ear, vertigo (which might be associated with loss of consciousness), vomiting, and, in many cases, progressive loss of hearing. The term is now used to include all cases of sudden vertigo accompanied by noises in the ear and deafness. The frequency of vertigo with ear symptoms is striking. Thus, of 106 cases noted by Gowers, in which there was definite vertigo, in 94 ear symptoms were present, either tinnitus or deafness or both.

Symptoms.—The attack usually sets in suddenly with a buzzing noise in the ears and the patient feels as if he was reeling or staggering. He may feel himself to be reeling, or the objects about him may seem to be turning, or the phenomena may be combined. The attack is often so abrupt that the patient falls, though, as a rule, he has time to steady himself by grasping some neighboring object. There may be slight but transient loss of consciousness. In a few minutes, or even less, the vertigo passes off and the patient becomes pale and nauseated, a clammy sweat breaks out on the face, and vomiting may follow.

The deafness, which is always of a nervous character, may be in only one ear and is never complete. As a rule, the patients have no affection of the middle ear. The tinnitus is described as either a roaring or a throbbing sound. Ocular symptoms may be present; thus, jerking of the eyeballs or nystagmus may develop during the attack, or diplopia.

Labyrinthine vertigo is paroxysmal, coming on at irregular intervals. Sometimes weeks or months may elapse between the attacks; in other cases there may be several attacks in a day. The disease rarely occurs in young persons, is most frequent after the fortieth year, and is more common in men than in women.

The pathology of the disease has been much discussed, and there are many theories. It seems to be tolerably certain at present that the disturbances of equilibrium, including the vertigo, are dependent upon a disturbance of the functions of the vestibular nerve or of the organs with which this nerve is connected, either in its peripheral distribution or by means of its central connection. The auditory symptoms often accompanying it are doubtless always due to involvement of the cochlear nerve or its peripheral or central connections.

Diagnosis.—The combination of tinnitus with giddiness, with or without gastric disturbance, is sufficient to establish a diagnosis. There are other forms of vertigo from which it must be distinguished. The form

Look up Menieres original description.

The following grouping of the cases has been made by H. Parkes Weber (1) The apopectic form, due to haemorrhage into the labyrinth, as in leukaemia, followed as a rule by complete deafness in one or both ears. (2) The cases associated with progressive inflammatory disease of the labyrinth. (3) Associated with organic changes in the auditory nerves, as in tumors, sometimes in tabes, and in cases of aural vertigo associated with facial paralysis on one side. (4) Cases in which a paroxysm of epilepsy is preceded by an auditory aura. (5) The ~~mild~~ moderate attacks which are associated with the various middle ear affections, with wax in the meatus, with violent syringing of the ears, etc., all of which are probably due to increase in the intra-labyrinthine pressure. There are other instances in which Menieres symptoms may be due to temporary excessive increase in the perilymph, possibly of angio-neurotic character.

known as gastric vertigo, which is associated with dyspepsia and occurs most commonly in persons of middle age, is, as a rule, readily distinguished by the absence of tinnitus or evidences of disturbance in the function of the auditory nerve. This variety of vertigo is much less common than Trousseau's description would lead us to believe. It is important to note the close connection of vertigo with ocular defects.

The cardio-vascular vertigo, one of the most common forms, occurs in cases of valvular disease, particularly aortic insufficiency, and as frequently in arterio-sclerosis.

Endemic Paralytic Vertigo.—In parts of Switzerland and France there is a remarkable form of vertigo described by Gerlier, which is characterized by attacks of paretic weakness of the extremities, falling of the eyelids, remarkable depression, but with retention of consciousness. It occurs also in northern Japan, where Miura says it develops paroxysmally among the farm laborers of both sexes and all ages. It is known there as *kubisagari*.

Aural vertigo must be carefully distinguished from attacks of *petit mal*, or, indeed, of definite epilepsy. It is rare in *petit mal* to have noises in the ear or actual giddiness, but in the aura preceding an epileptic attack the patient may feel giddy. Giddiness and transient loss of consciousness may be associated with organic disease of the brain, more particularly with tumor. Vomiting also may be present. A careful investigation of the symptoms will usually lead to a correct diagnosis.

The outlook in Ménière's disease is uncertain. While many cases recover completely, in others deafness results and the attacks recur at shorter intervals. In aggravated cases the patient constantly suffers from vertigo and may even be confined to his bed.

Treatment.—Bromide of potassium, in 20-grain doses three times a day, is sometimes beneficial. If there is a history of syphilis, the iodide should be administered. The salicylates are recommended, and Charcot advises quinine to cinchonism. In cases in which there is increase in the arterial tension, nitroglycerin may be given, at first in very small doses, but increasing gradually. It is not specially valuable in Ménière's disease, but in the cases of giddiness in middle-aged men and women associated with arterio-sclerosis it sometimes acts very satisfactorily. Correction of errors of refraction is sometimes followed by prompt relief of the vertigo.

Glosso-pharyngeal Nerve (*Nervus glossopharyngeus*).

The ninth nerve contains both motor and sensory fibres and is also a nerve of the special sense of taste to the tongue. It supplies, by its motor branches, the stylo-pharyngeus and the middle constrictor of the pharynx. The sensory fibres are distributed to the upper part of the pharynx.

Symptoms.—Of nuclear disturbance we know very little. The pharyngeal symptoms of bulbar paralysis are probably associated with involvement of the nuclei of this nerve. Lesion of the nerve trunk itself is rare, but it may be compressed by tumors or involved in meningitis. Disturbance of the sense of taste may result from loss of function of this nerve, in which case it is chiefly in the posterior part of the tongue and soft pal-

ate. Gowers, however, states that there is no case on record in which loss of taste in these regions has been produced by disease of the roots of the glosso-pharyngeal; whereas, on the other hand, disease of the root of the fifth nerve may cause loss of taste on the back as well as the front of the tongue, as if the taste fibres of the glosso-pharyngeal came from the fifth.

The general disturbances of the sense of taste may here be briefly referred to. Loss of the sense of taste—*ageusia*—may be caused by disturbance of the peripheral end organs, as in affections of the mucosa of the tongue. This is very common in the dry tongue of fever or the furred tongue of dyspepsia, under which circumstances, as the saying is, everything tastes alike. Strong irritants too, such as pepper, tobacco, or vinegar, may dull or diminish the sense of taste. Complete loss may be due to involvement of the nerves either in their course or in the centres. Disturbance in the sense of taste is most commonly seen in involvement of the fifth nerve, and it may be that this nerve alone subserves the function. Perversion of the sense of taste—*parageusis*—is rarely found, except as an hysterical manifestation and in the insane. Increased sensitiveness is still more rare. There are occasional subjective sensations of taste, occurring as an aura in epilepsy or as part of the hallucinations in the insane.

To test the sense of taste the patient's eyes should be closed and small quantities of various substances applied. The sensation should be perceived before the tongue is withdrawn. The following are the most suitable tests: For bitter, quinine; for sweetness, a strong solution of sugar or saccharin; for acidity, vinegar; and for the saline test, common salt. One of the most important tests is the feeble galvanic current, which gives the well-known metallic taste.

Pneumogastric Nerve (*Nervus vagus*).

The tenth nerve has an important and extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach. The nerve may be involved at its nucleus along with the spinal accessory and the hypoglossal, forming what is known as bulbar paralysis. It may be compressed by tumors or aneurism, or in the exudation of meningitis, simple or syphilitic. In its course in the neck the trunk may be involved by tumors or in wounds. It has been tied in ligature of the carotid, and has been cut in the removal of deep-seated tumors. The trunk may be attacked by neuritis.

The affections of the vagus are best considered in connection with the distribution of the separate nerves.

(*a*) **Pharyngeal Branches.**—In combination with the glosso-pharyngeal the branches from the vagus form the pharyngeal plexus, from which the muscles and mucosa of the pharynx are supplied. In *paralysis* due to involvement of this either in the nuclei, as in bulbar paralysis, or in the course of the nerve, as in diphtheritic neuritis, there is difficulty in swallowing and the food is not passed on into the œsophagus. If the nerve on one side only is involved, the deglutition is not much impaired. In these

cases the particles of food frequently pass into the larynx, and, when the soft palate is involved, into the posterior nares.

Spasm of the pharynx is always a functional disorder, usually occurring in hysterical and nervous people. Gowers mentions a case of a gentleman who could not eat unless alone, on account of the inability to swallow in the presence of others from spasm of the pharynx. This spasm is a well-marked feature in hydrophobia, and I have seen it in a case of pseudo-hydrophobia.

(*b*) **Laryngeal Branches.**—The superior laryngeal nerve supplies the mucous membrane of the larynx above the cords and the crico-thyroid muscle. The inferior or recurrent laryngeal curves around the arch of the aorta on the left side and the subclavian artery on the right, passes along the trachea and supplies the mucosa below the cords and all the muscles of the larynx except the crico-thyroid and the epiglottidean. Experiments have shown that these motor nerves of the pneumogastric are all derived from the spinal accessory. The remarkable course of the recurrent laryngeal nerves renders them liable to pressure by tumors within the thorax, particularly by aneurism. The following are the most important forms of paralysis:

(1) *Bilateral Paralysis of the Abductors.*—In this condition, the posterior crico-arytenoids are involved and the glottis is not opened during inspiration. The cords may be close together in the position of phonation, and during inspiration may be brought even nearer together by the pressure of air, so that there is only a narrow chink through which the air whistles with a noisy stridor. This dangerous form of laryngeal paralysis occurs occasionally as a result of cold, or may follow a laryngeal catarrh. The posterior muscles have been found degenerated when the others were healthy. The condition may be produced by pressure upon both vagi, or upon both recurrent nerves. As a central affection it occurs in tabes and bulbar paralysis, but may be seen also in hysteria. The characteristic symptoms are inspiratory stridor with unimpaired phonation. Possibly, as Gowers suggests, many cases of so-called hysterical spasm of the glottis are in reality abductor paralysis.

(2) *Unilateral Abductor Paralysis.*—This frequently results from the pressure of tumors or involvement of one recurrent nerve. Aneurism is by far the most common cause, though on the right side the nerve may be involved in thickening of the pleura. The symptoms are hoarseness or roughness of the voice, such as is so common in aneurism. Dyspnœa is not often present. The cord on the affected side does not move in inspiration. Subsequently the adductors may also become involved, in which case the phonation is still more impaired.

(3) *Adductor Paralysis.*—This results from involvement of the lateral crico-arytenoid and the arytenoid muscle itself. It is common in hysteria, particularly of women, and causes the hysterical aphonia, which may come on suddenly. It may result from catarrh of the larynx or from overuse of the voice. In laryngoscopic examination it is seen, on attempt at phonation, that there is no power to bring the cords together. In this connection the following table from Gowers' work will be found valuable to the student:

Symptoms.	Signs.	Lesion.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the Muscles of the Larynx.—In this the adductor muscles are involved. It is not an uncommon affection in children, and has already been referred to as laryngismus stridulus. Paroxysmal attacks of laryngeal spasm are rare in the adult, but cases are described in which the patient, usually a young girl, wakes at night in an attack of intense dyspnœa, which may persist long enough to produce cyanosis. Liveing states that they may replace attacks of migraine. They occur in a characteristic form in locomotor ataxia, forming the so-called laryngeal crises. There is a condition known as spastic aphonia, in which, when the patient attempts to speak, phonation is completely prevented by a spasm.

Disturbance of the sensory nerves of the larynx is rare.

Anæsthesia may occur in bulbar paralysis and in diphtheritic neuritis—a serious condition, as portions of food may enter the windpipe. It is usually associated with dysphagia and is sometimes present in hysteria. Hyperæsthesia of the larynx is rare.

(*c*) **Cardiac Branches.**—The cardiac plexus is formed by the union of branches of the vagi and of the sympathetic nerves. The vagus fibres subserve motor, sensory, and probably trophic functions.

(1) *Motor.*—The fibres which inhibit, control, and regulate the cardiac action pass in the vagi. Irritation may produce slowing of the action. Czermak could slow or even arrest the heart's action for a few beats by pressing a small tumor in his neck against one pneumogastric nerve, and it is said

that the same can be produced by forcible bilateral pressure on the carotid canal. There are instances in which persons appear to have had voluntary control over the action of the heart. Cheyne mentions the case of Colonel Townshend, "who could die or expire when he pleased, and yet by an effort or somehow come to life again, which it seems he had sometimes tried before he had sent for us." Retardation of the heart's action has also followed accidental ligature of one vagus. Irritation at the nuclei may also be accompanied with a neurosis of this nerve. On the other hand, when there is complete paralysis of the vagi, the inhibitory action may be abolished and the acceleratory influences have full sway. The heart's action is then greatly increased. This is seen in some instances of diphtheritic neuritis and in involvement of the nerve by tumors, or its accidental removal or ligature. Complete loss of function of one vagus may, however, not be followed by any symptoms.

(2) *Sensory* symptoms on the part of the cardiac branches are very varied. Normally, the heart's action proceeds regularly without the participation of consciousness, but the unpleasant feelings and sensations of palpitation and pain are conveyed to the brain through this nerve. How far the fibres of the pneumogastric are involved in angina it is impossible to say. The various disturbances of sensation are described under the cardiac neuroses.

(*d*) **Pulmonary Branches.**—We know very little of the pulmonary branches of the vagi. The motor fibres are stated to control the action of the bronchial muscles, and it has long been held that asthma may be a neurosis of these fibres. The various alterations in the respiratory rhythm are probably due more to changes in the centre than in the nerves themselves.

(*e*) **Gastric and Œsophageal Branches.**—The muscular movements of these parts are presided over by the vagi and vomiting is induced through them, usually reflexly, but also by direct irritation, as in meningitis. Spasm of the œsophagus generally occurs with other nervous phenomena. Gastralgia may sometimes be due to cramp of the stomach, but is more commonly a sensory disturbance of this nerve, due to direct irritation of the peripheral ends, or is a neuralgia of the terminal fibres. Hunger is said to be a sensation aroused by the pneumogastric, and some forms of nervous dyspepsia probably depend upon disturbed function of this nerve. The severe gastric crises which occur in locomotor ataxia are due to central irritation of the nuclei. Some describe exophthalmic goitre under lesions of the vagi.

Spinal Accessory Nerve (*Nervus accessorius*).

Paralysis.—The smaller or internal part of this nerve joins the vagus and is distributed through it to the laryngeal muscles. The larger external part is distributed to the sterno-mastoid and trapezius muscles.

The nuclei of the nerve, particularly of the accessory part, may be involved in bulbar paralysis. The nuclei of the external portion, situated as they are in the cervical cord, may be attacked in progressive degeneration of the motor nuclei of the cord. The nerve may be involved in the exudation of meningitis, or be compressed by tumors, or in caries. The

symptoms of paralysis of the accessory portion which joins the vagus have already been given in the account of the palsy of the laryngeal branches of the pneumogastric. Disease or compression of the external portion is followed by paralysis of the sterno-mastoid and of the trapezius on the same side. In paralysis of one sterno-mastoid, the patient rotates the head with difficulty to the opposite side, but there is no torticollis, though in some cases the head is held obliquely. As the trapezius is supplied in part from the cervical nerves, it is not completely paralyzed, but the portion which passes from the occipital bone to the acromion is functionless. The paralysis of the muscle is well seen when the patient draws a deep breath or shrugs the shoulders. The middle portion of the trapezius is also weakened, the shoulder droops a little, and the angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is impaired, for the trapezius does not fix the scapula as a point from which the deltoid can work.

In progressive muscular atrophy we sometimes see bilateral paralysis of these muscles. Thus, if the sterno-mastoids are affected, the head tends to fall back; when the trapezii are involved, it falls forward, a characteristic attitude of the head in many cases of progressive muscular atrophy. Gowers suggests that lesions of the accessory in difficult labor may account for those cases in which during the first year of life the child has great difficulty in holding up the head. In children this drooping of the head is an important symptom in cervical meningitis, the result of caries.

The *treatment* of the condition depends much upon the cause. In the central nuclear atrophy but little can be done. In paralysis from pressure the symptoms may gradually be relieved. The paralyzed muscles should be stimulated by electricity and massage.

Accessory Spasm.—(*Torticollis; Wryneck.*)—The forms of spasm affecting the cervical muscles are best considered here, as the muscles supplied by the accessory are chiefly, though not solely, responsible for the condition. The following forms may be described in this section:

(*a*) *Congenital Torticollis.*—This condition, also known as fixed torticollis, depends upon the shortening and atrophy of the sterno-mastoid on one side. It occurs in children and may not be noticed for several years on account of the shortness of the neck, the parents often alleging that it has only recently come on. It affects the right side almost exclusively. A remarkable circumstance in connection with it is the existence of facial asymmetry noted by Wilks, which appears to be an essential part of this congenital form. It occurred in 6 cases reported by Golding-Bird. In congenital wryneck the sterno-mastoid is shortened, hard and firm, and in a condition of more or less advanced atrophy. This must be distinguished from the local thickening in the sterno-mastoid due to rupture, which may occur at the time of birth and produce an induration or muscle callus. Although the sterno-mastoid is almost always affected, there are rare cases in which the fibrous atrophy affects the trapezius. This form of wryneck in itself is unimportant, since it is readily relieved by tenotomy, but Golding-Bird states that the facial asymmetry persists, or indeed may, as shown by photographs in my case, become more evident. With reference

to the pathology of the affection, Golding-Bird concludes that the facial asymmetry and the torticollis are integral parts of one affection which has a central origin and is the counterpart in the head and neck of infantile paralysis with talipes in the foot.

(*b*) *Spasmodic Wryneck.*—Two varieties of this spasm occur, the tonic and the clonic, which may alternate in the same case; or, as is most common, they are separate and remain so from the outset. The disease is most frequent in adults and, according to Gowers, more common in females. In this country it is certainly more frequent in males. Of the 8 or 10 cases which came under my observation in Montreal and Philadelphia, all were males. In females it may be an hysterical manifestation. There may be a marked neurotic family history, but it is usually impossible to fix upon any definite etiological factor. Some cases have followed cold; others a blow.

The *symptoms* are well defined. In the tonic form the contracted sterno-mastoid draws the occiput toward the shoulder of the affected side; the chin is raised, and the face rotated to the other shoulder. The sterno-mastoid may be affected alone or in association with the trapezius. When the latter is implicated the head is depressed still more toward the same side. In long-standing cases these muscles are prominent and very rigid. There may be some curvature of the spine, the convexity of which is toward the sound side. The cases in which the spasm is clonic are much more distressing and serious. The spasm is rarely limited to a single muscle. The sterno-mastoid is almost always involved and rotates the head so as to approximate the mastoid process to the inner end of the clavicle, turning the face to the opposite side and raising the chin. When with this the trapezius is affected, the depression of the head toward the same side is more marked. The head is drawn somewhat backward; the shoulder, too, is raised by its action. According to Gowers, the splenius is associated with the sterno-mastoid about half as frequently as the trapezius. Its action is to incline the head and rotate it slightly toward the same side. Other muscles may be involved, such as the scalenus and platysma myoides; and in rare cases the head may be rotated by the deep cervical muscles, the rectus and obliquus. There are cases in which the spasm is bilateral, causing a backward movement—the retro-collic spasm. This may be either tonic or clonic, and in extreme cases the face is horizontal and looks upward.

These clonic contractions may come on without warning, or be preceded for a time by irregular pains or stiffness of the neck. The jerking movements recur every few moments, and it is impossible to keep the head still for more than a minute or two. In time the muscles undergo hypertrophy and may be distinctly larger on one side than the other. In some cases the pain is considerable; in others there is simply a feeling of fatigue. The spasms cease during sleep. Emotion, excitement, and fatigue increase them. The spasm may extend from the muscles of the neck and involve those of the face or of the arms.

The disease varies much in its course. Cases occasionally get well, but the great majority of them persist, and, even if temporarily relieved, the disease frequently recurs. The affection is usually regarded as a functional

neurosis, but it is possibly due to disturbance of the cortical centres presiding over the muscles.

Treatment.—Temporary relief is sometimes obtained; a permanent cure is exceptional. Various drugs have been used, but rarely with benefit. Occasionally, large doses of bromide will lessen the intensity of the spasm. Morphia, subcutaneously, has been successful in some reported cases, but there is the great danger of establishing the morphia habit. Galvanism may be tried. Counter-irritation is probably useless. Fixation of the head mechanically can rarely be borne by the patient. These obstinate cases fall ultimately into the hands of the surgeon, and the operations of stretching, division, and excision of the accessory nerve and division of the muscles have been tried. The last does not check the spasm, and may aggravate the symptoms. Temporary relief may follow, but, as a rule, the condition returns. Risien Russell thinks that resection of the posterior branches of the upper cervical nerves is most likely to give relief, and this has been done by Keen and others.

(*c*) The *nodding spasm* of children may here be mentioned as involving chiefly the muscles innervated by the accessory nerve. It may be a simple trick, a form of habit spasm, or a phenomenon of epilepsy (E. nutans), in which case it is associated with transient loss of consciousness. A similar nodding spasm may occur in older children. In women it sometimes occurs as an hysterical manifestation, commonly as part of the so-called salaam convulsion.

Hypoglossal Nerve.

This is the motor nerve of the tongue and for most of the muscles attached to the hyoid bone. Its cortical centre is probably the lower part of the anterior central gyrus.

Paralysis.—(1) *Cortical Lesion.*—The tongue is often involved in hemiplegia, and the paralysis may result from a lesion of the cortex itself, or of the fibres as they pass to the medulla. It does not occur alone and is considered with hemiplegia. There is this difference, however, between the cortical and other forms, that the muscles on both sides of the tongue may be more or less affected but do not waste, nor are their electrical reactions disturbed.

(2) *Nuclear* and *infra-nuclear* lesions of the hypoglossal result from slow progressive degeneration, as in bulbar paralysis or in locomotor ataxia; occasionally there is acute softening from obstruction of the vessels. The nuclei of both nerves are usually affected together, but may be attacked separately. Trauma and lead poisoning have also been assigned as causes. The fibres may be damaged by a tumor, and at the base by meningitis; or the nerve is sometimes involved in the condylar foramen by disease of the skull. It may be involved in its course in a scar, as in Birkett's case, or compressed by a tumor in the parotid region, as in a case at present under my care. As a result, there is loss of function in the nerve fibres and the tongue undergoes atrophy on the affected side. It is protruded toward the paralyzed side and may show fibrillary twitching.

The *symptoms* of involvement of one hypoglossal, either at its centre

or in its course, are those of unilateral paralysis and atrophy of the tongue. When protruded, it is pushed toward the affected side, and there are fibrillary twitchings. The atrophy is usually marked and the mucous membrane on the affected side is thrown into folds. Articulation is not much impaired in the unilateral affection. There is a remarkable triad of symptoms, to which Hughlings Jackson first called attention—unilateral hemi-atrophy of the tongue, loss of power in the palate muscle, with paralysis of the larynx on the same side. When the disease is bilateral, the tongue lies almost motionless in the floor of the mouth; it is atrophied, and cannot be protruded. Speech and mastication are extremely difficult and deglutition may be impaired. If the seat of the disease is above the nuclei, there may be little or no wasting. The condition is seen in progressive bulbar paralysis and occasionally in progressive muscular atrophy.

The *diagnosis* is readily made and the situation of the lesion can usually be determined, since when supra-nuclear there is associated hemiplegia and no wasting of the muscles of the tongue. Nuclear disease is only occasionally unilateral; most commonly bilateral and part of a bulbar paralysis. It should be borne in mind that the fibres of the hypoglossal may be involved within the medulla after leaving their nuclei. In such a case there may be paralysis of the tongue on one side and paralysis of the limbs on the opposite side, and the tongue, when protruded, is pushed toward the sound side.

Spasm.—This rare affection may be unilateral or bilateral. It is most frequently a part of some other convulsive disorder, such as epilepsy, chorea, or spasm of the facial muscles. In some cases of stuttering, spasm of the tongue precedes the explosive utterance of the words. It may occur in hysteria, and is said to follow reflex irritation in the fifth nerve. The most remarkable cases are those of paroxysmal clonic spasm, in which the tongue is rapidly thrust in and out, as many as forty or fifty times a minute. In the case reported by Gowers the attacks occurred during sleep and continued for a year and a half. The spasm is usually bilateral. Wendt has reported a case in which it was unilateral. The prognosis is usually good.

IV. DISEASES OF THE SPINAL NERVES.

Cervical Plexus.

(1) **Occipito-cervical Neuralgia.**—This involves the nerve territory supplied by the second, the occipitalis major and minor, and the auricularis magnus nerves. The pains are chiefly in the back of the head and neck and in the ear. The condition may follow cold and is sometimes associated with stiffness of the neck or torticollis. Unless connected with it there exists disease of the bones or due to pressure of tumors, the outlook is usually good. There are tender points midway between the mastoid process and the spine and just above the parietal eminence, and between the sterno-mastoid and the trapezius. The affection may be due to direct pressure, in persons who carry very heavy loads on the neck.

(2) **Affections of the Phrenic Nerve.**—Paralysis may follow a lesion in the anterior horns at the level of the third and fourth cervical nerves, or may be due to compression of the nerve by tumors or aneurism. More rarely paralysis results from neuritis.

It may be part of a diphtheritic or lead palsy and is usually bilateral. When the diaphragm is paralyzed respiration is carried on by the intercostal and accessory muscles. When the patient is quiet and at rest little may be noticed, but the abdomen retracts in inspiration and is forced out in expiration. On exertion or even on attempting to move there may be dyspnœa. If the paralysis sets in suddenly there may be dyspnœa and lividity, which is usually temporary (W. Pasteur). Intercurrent attacks of bronchitis seriously aggravate the condition. Difficulty in coughing, owing to the impossibility of drawing a full breath, adds greatly to the danger of this complication, as the mucus accumulates in the tubes.

When the phrenic nerve is paralyzed on one side the paralysis may be scarcely noticeable, but careful inspection shows that the descent of the diaphragm is much less on the affected side.

The *diagnosis* of paralysis is not always easy, particularly in women, who habitually use this muscle less than men, and in whom the diaphragmatic breathing is less conspicuous. Immobility of the diaphragm is not uncommon, particularly in diaphragmatic pleurisy, in large effusions, and in extensive emphysema. The muscle itself may be degenerated and its power impaired.

Owing to the lessened action of the diaphragm, there is a tendency to accumulation of blood at the bases of the lungs, and there may be impaired resonance and signs of œdema. As a rule, however, the paralysis is not confined to this muscle, but is part of a general neuritis or an anterior polio-myelitis, and there are other symptoms of value in determining its presence. The outlook is usually serious. Pasteur states that of 15 cases following diphtheria, only 8 recovered. The treatment is that of the neuritis or polio-myelitis with which it is associated.

Hiccough.—Here may, perhaps, best be considered this remarkable symptom, caused by intermittent, sudden contraction of the diaphragm. The mechanism, however, is complex, and while the afferent impressions to the respiratory centre may be peripheral or central, the efferent are distributed through the phrenic nerve to the diaphragm, causing the intermittent spasm, and through the laryngeal branches of the vagus to the glottis, causing sudden closure as the air is rapidly inspired.

Obstinate hiccough is one of the most distressing of all symptoms, and may tax to the uttermost the resources of the physician. W. Langford Symes in a recent study groups the cases into:

(*a*) *Inflammatory,* seen particularly in affections of the abdominal viscera, gastritis, peritonitis, hernia, internal strangulation, appendicitis, suppurative pancreatitis, and in the severe forms of typhoid fever.

(*b*) *Irritative,* as in the direct stimulus of the diaphragm in the swallowing of very hot substances, local disease of the œsophagus near the diaphragm, and in many conditions of gastric and intestinal disorder, more particularly those associated with flatus.

Dear Doctor: Aurora, Ill.
July 12, 1900.

Nothing gave permanent relief from the case of functional hiccoughing of two months duration, till I empirically dilated the uterine cervix instrumentally without anaesthesia. Relief was immediate and permanent.

Yours truly,
C. F. Maxwell, M.D.

(*c*) *Specific*, or, perhaps more properly, *idiopathic*, in which no evident causes are present. In these cases there is usually some constitutional taint, as gout, diabetes, or chronic Bright's disease. I have seen several instances of obstinate hiccough in the later stages of chronic interstitial nephritis.

(*d*) *Neurotic*, cases in which the primary cause is in the nervous system; hysteria, epilepsy, shock, or cerebral tumors. Of these cases the hysterical are, perhaps, the most obstinate.

The treatment is often very unsatisfactory. Sometimes in the milder forms a sudden reflex irritation will check it at once. Readers of Plato's Symposium will remember that the physician Eryximachus recommended to Aristophanes, who had hiccough from eating too much, either to hold his breath (which for trivial forms of hiccough is very satisfactory) or to gargle with a little water; but if it still continued, "tickle your nose with something and sneeze; and if you sneeze once or twice even the most violent hiccough is sure to go." The attack must have been of some severity, as it is stated subsequently that the hiccough did not disappear until Aristophanes had resorted to the sneezing.

Ice, a teaspoonful of salt and lemon-juice, or salt and vinegar, or a teaspoonful of raw spirits may be tried. When the hiccough is due to gastric irritation, lavage is sometimes promptly curative. I saw a case of a week's duration cured by a hypodermic injection of gr. $\frac{1}{8}$ of apomorphia. In obstinate cases the various antispasmodics have been used in succession. Pilocarpine has been recommended. One has sometimes to resort to hypodermics of morphia, or to inhalations of chloroform. The nitrite of amyl and nitroglycerin have been beneficial in some cases. Galvanism over the phrenic nerve, or pressure on the nerves, applied between the heads of the sterno-cleido-mastoid muscles may be used. ~~In the very severe forms all these measures may prove futile~~.

Brachial Plexus.

(1) **Combined Paralysis.**—The plexus may be involved in the supraclavicular region by compression of the nerve trunks as they leave the spine, or by tumors and other morbid processes in the neck. Below the clavicle lesions are more common and result from injuries following dislocation or fracture, sometimes from neuritis. The most common cause of lesion of the brachial plexus is luxation of the humerus, particularly the subcoracoid form. If the dislocation is quickly reduced the symptoms are quite transient, and disappear in a few days. In severe cases all the branches of the plexus, or only one or two, may be involved. The most serious cases are those in which the dislocation is undetected or unreduced for some time, when the prolonged pressure on the nerves may cause complete and permanent paralysis of the arm. The muscles waste, the reaction of degeneration is present, and trophic changes in the skin are apt to occur. The medico-legal bearings of these cases are important, and may be thus briefly summarized: Direct injury, as by a fall or blow on the shoulder, resulting in great bruising of the nerves without dislocation, is occasionally followed by complete paralysis of the arm. A dislocation may be set immediately and

yet the lesion of the brachial plexus may be such as to cause permanent paralysis of the nerves. The dislocation may be reduced and the joint in subsequent movements slips out again. It has happened that by the time the surgeon sees the patient again, the damage has become irreparable.

Injuries and blows on the neck may cause partial paralysis of the arm, involving the deltoid, supraspinatus, infraspinatus, biceps, brachialis anticus, and the supinator. The injury may occur to the child during delivery.

A primary neuritis of the brachial plexus is rare. More commonly the process is an ascending neuritis from a lesion of a peripheral branch, involving first the radial or ulnar nerves, and spreading upward to the plexus, producing gradually complete loss of power in the arm.

(2) Lesions of Individual Nerves of the Plexus.—(*a*) *Long Thoracic Nerve* (*Serratus Palsy*).—This occurs chiefly in men. The nerve is injured in the posterior triangle of the neck, usually by direct pressure in the carrying of loads; cold may cause neuritis. It may be involved also in progressive muscular atrophy and in polio-myelitis anterior. When paralyzed the scapula on the affected side looks winged, which results from the projection of the angle and posterior border. This is particularly noticeable when the arm is moved forward, when the serratus no longer holds the scapula against the thorax. It is a well-defined and readily recognized form of paralysis. The onset is associated with, sometimes preceded by, neuralgic pains. The course is dubious, and many months may elapse before there is any improvement.

(*b*) *Circumflex Nerve.*—This supplies the deltoid and the teres minor. The nerve is apt to be involved in injuries, in dislocations, bruising by a crutch, or sometimes by extension of inflammation from the joint. Occasionally the paralysis arises from a pressure neuritis during an illness. As a consequence of loss of power in the deltoid, the arm cannot be raised. The wasting is usually marked and changes the shape of the shoulder. Sensation may also be impaired in the skin over the muscle. The joint may be relaxed and there may be a distinct space between the head of the humerus and the acromion. In other instances the ligaments are thickened, and a condition not unlike ankylosis may be produced, but which is readily distinguished on moving the arm.

(*c*) *Musculo-spiral Paralysis; Radial Paralysis.*—This is one of the most common of peripheral palsies, and results from the exposed position of the musculo-spiral nerve. It is often bruised in the use of the crutch, by injuries of the arm, blows, or fractures. It is frequently injured when a person falls asleep with the arm over the back of a chair, or by pressure of the body upon the arm when a person is sleeping on a bench or on the ground. It may be paralyzed by sudden violent contraction of the triceps. It is sometimes involved in a neuritis from cold, but this is uncommon in comparison with other causes. In the subcutaneous injection of ether the nerve may be accidentally struck and temporarily paralyzed. The paralysis of lead poisoning is the result of involvement of certain branches of this nerve.

A lesion when high up involves the triceps, the brachialis anticus, and

the supinator longus, as well as the extensors of the wrist and fingers. Naturally, in lesions just above the elbow the arm muscles and the supinator longus are spared. The most characteristic feature of the paralysis is the wrist-drop and the inability to extend the first phalanges of the fingers and thumb. In the pressure palsies the supinators are usually involved and the movements of supination cannot be accomplished. The sensations may be impaired, or there may be marked tingling, but the loss of sensation is rarely so pronounced as that of motion.

The affection is readily recognized, but it is sometimes difficult to say upon what it depends. The sleep and pressure palsies are, as a rule, unilateral and involve the supinator longus. The paralysis from lead is bilateral and the supinators are unaffected. Bilateral wrist-drop is a very common symptom in many forms of multiple neuritis, particularly the alcoholic; but the mode of onset and the involvement of the legs and arms are features which make the diagnosis easy. The duration and course of the musculo-spiral paralyses are very variable. The pressure palsies may disappear in a few days. Recovery is the rule, even when the affection lasts for many weeks. The electrical examination is of importance in the prognosis, and the rules laid down under paralysis of the facial nerve hold good here.

The treatment is that of neuritis.

(*d*) *Ulnar Nerve.*—The motor branches supply the ulnar halves of the deep flexor of the fingers, the muscles of the little finger, the interossei, the adductor and the inner head of the short flexor of the thumb, and the ulnar flexor of the wrist. The sensory branches supply the ulnar side of the hand—two and a half fingers on the back, and one and a half fingers on the front. Paralysis may result from pressure, usually at the elbow-joint, although the nerve is here protected. Possibly the neuritis in the ulnar nerve in some cases of acute illness may be due to this cause. Gowers mentions the case of a lady who twice had ulnar neuritis after confinement. Owing to paralysis of the ulnar flexor of the wrist, the hand moves toward the radial side; adduction of the thumb is impossible; the first phalanges cannot be flexed, and the others cannot be extended. In long-standing cases the first phalanges are overextended and the others strongly flexed, producing the claw-hand; but this is not so marked as in the progressive muscular atrophy. The loss of sensation corresponds to the sensory distribution just mentioned.

(*e*) *Median Nerve.*—This supplies the flexors of the fingers except the ulnar half of the deep flexors, the abductor and the flexors of the thumb, the two radial lumbricales, the pronators, and the radial flexor of the wrist. The sensory fibres supply the radial side of the palm and the front of the thumb, the first two fingers and half the third finger, and the dorsal surfaces of the same three fingers.

This nerve is seldom involved alone. Paralysis results from injury and occasionally from neuritis. The signs are inability to pronate the forearm beyond the mid-position. The wrist can only be flexed toward the ulnar side; the thumb cannot be opposed to the tips of fingers. The second phalanges cannot be flexed on the first; the distal phalanges of the first

and second fingers cannot be flexed; but in the third and fourth fingers this action can be performed by the ulnar half of the flexor profundus. The loss of sensation is in the region corresponding to the sensory distribution already mentioned. The wasting of the thumb muscles, which is usually marked in this paralysis, gives to it a characteristic appearance.

Lumbar and Sacral Plexuses.

The *lumbar plexus* is sometimes involved in growths of the lymph-glands, in psoas abscess, and in disease of the bones of the vertebræ. Of its branches the *obturator nerve* is occasionally injured during parturition. When paralyzed the power is lost over the adductors of the thigh and one leg cannot be crossed over the other. Outward rotation is also disturbed. The *anterior crural nerve* is sometimes involved in wounds or in dislocation of the hip-joint, less commonly during parturition, and sometimes by disease of the bones and in psoas abscess. The special symptoms of affection of this nerve are paralysis of the extensors of the knee with wasting of the muscles, anæsthesia of the antero-lateral parts of the thigh and of the inner side of the leg to the big toe. This nerve is sometimes involved early in growths about the spine, and there may be pain in its area of distribution. Loss of the power of abducting the thigh results from paralysis of the *gluteal nerve*, which is distributed to the gluteus, medius, and minimus muscles.

The *sacral plexus* is frequently involved in tumors and inflammations within the pelvis and may be injured during parturition. Neuritis is common, usually an extension from the sciatic nerve.

Of the branches, the *sciatic nerve*, when injured at or near the notch, causes paralysis of the flexors of the legs and the muscles below the knee, but injury below the middle of the thigh involves only the latter muscles. There is also anæsthesia of the outer half of the leg, the sole, and the greater portion of the dorsum of the foot. Wasting of the muscles frequently follows, and there may be trophic disturbances. In paralysis of one sciatic the leg is fixed at the knee by the action of the quadriceps extensor and the patient is able to walk.

Paralysis of the *small sciatic nerve* is rarely seen. The gluteus maximus is involved and there may be difficulty in rising from a seat. There is a strip of anæsthesia along the back of the middle third of the thigh.

External Popliteal Nerve.—Paralysis involves the peronæi, the long extensor of the toes, tibialis anticus, and the extensor brevis digitorum. The ankle cannot be flexed, resulting in a condition known as foot-drop, and as the toes cannot be raised the whole leg must be lifted, producing the characteristic *steppage* gait seen in so many forms of peripheral neuritis. In long-standing cases the foot is permanently extended and there is wasting of the anterior tibial and peroneal muscles. The loss of sensation is in the outer half of the front of the leg and on the dorsum of the foot.

Internal Popliteal Nerve.—When paralyzed, plantar flexion of the foot and flexion of the toes are impossible. The foot cannot be adducted, nor can the patient rise on tiptoe. In long-standing cases talipes calcaneus

follows and the toes assume a claw-like position from secondary contracture, due to overextension of the proximal and flexion of the second and third phalanges.

Sciatica.

This is, as a rule, a neuritis either of the sciatic nerve or of its cords of origin. It may in some instances be a functional neurosis or neuralgia.

It occurs most commonly in adult males. A history of rheumatism or of gout is present in many cases. Exposure to cold, particularly after heavy muscular exertion, or a severe wetting are not uncommon causes. Within the pelvis the nerves may be compressed by large ovarian or uterine tumors, by lymphadenomata, by the fœtal head during labor; occasionally lesions of the hip-joint induce a secondary sciatica. The condition of the nerve has been examined in a few cases, and it has often been seen in the operation of stretching. It is, as a rule, swollen, reddened, and in a condition of interstitial neuritis. The affection may be most intense at the sciatic notch or in the nerve about the middle of the thigh.

Of the *symptoms*, pain is the most constant and troublesome. The onset may be severe, with slight pyrexia, but, as a rule, it is gradual, and for a time there is only slight pain in the back of the thigh, particularly in certain positions or after exertion. Soon the pain becomes more intense, and instead of being limited to the upper portion of the nerve, extends down the thigh, reaching the foot and radiating over the entire distribution of the nerve. The patient can often point out the most sensitive spots, usually at the notch or in the middle of the thigh; and on pressure these are exquisitely painful. The pain is described as gnawing or burning, and is usually constant, but in some instances is paroxysmal, and often worse at night. On walking it may be very great; the knee is bent and the patient treads on the toes, so as to relieve the tension on the nerve. In protracted cases there may be much wasting of the muscles, but the reaction of degeneration can seldom be obtained. In these chronic cases cramp may occur and fibrillar contractions. Herpes may develop, but this is unusual. In rare instances the neuritis ascends and involves the spinal cord.

The duration and course are extremely variable. As a rule it is an obstinate affection, lasting for months, or even, with slight remissions, for years. Relapses are not uncommon, and the disease may be relieved in one nerve only to appear in the other. In the severer forms the patient is bedridden, and such cases prove among the most distressing and trying which the physician is called upon to treat.

In the *diagnosis* it is important, in the first place, to determine whether the disease is primary, or secondary to some affection of the pelvis or of the spinal cord. A careful rectal examination should be made, and, in women, pelvic tumor should be excluded. Lumbago may be confounded with it. Affections of the hip-joint are easily distinguished by the absence of tenderness in the course of the nerve and the sense of pain on movement of the hip-joint or on pressure in the region of the trochanter. There are instances of sacro-iliac disease in which the patient complains of pain in the upper part of the thigh, which may sometimes radiate; but careful

examination will readily distinguish between the affections. Pressure on the nerve trunks of the cauda equina, as a rule, causes bilateral pain and disturbances of sensation, and, as double sciatica is rare, these circumstances always suggest lesion of the nerve roots. Between the severe lightning pains of tabes and sciatica the differences are usually well defined.

Treatment.—The pelvic organs should be carefully and systematically examined. Constitutional conditions, such as rheumatism and gout, should receive appropriate treatment. In a few cases with pronounced rheumatic history, which come on acutely with fever, the salicylates seem to do good. In other instances they are quite useless. If there is a suspicion of syphilis, the iodide of potassium should be employed, and in gouty cases salines.

Rest in bed with fixation of the limb by means of a long splint is a most valuable method of treatment in many cases, one upon which Weir Mitchell has specially insisted. I have known it to relieve, and in some instances to cure, obstinate and protracted cases which had resisted all other treatment. Hydrotherapy is sometimes satisfactory, particularly the warm baths or the mud baths. Many cases are relieved by a prolonged residence at one of the thermal springs.

Antipyrin, antifebrin, and quinine, are of doubtful benefit.

Local applications are more beneficial. The hot iron or the thermocautery or blisters relieve the pain temporarily. Deep injections into the nerves give great relief and may be necessary for the pain. It is best to use cocaine at first, in doses of from an eighth to a quarter of a grain. If the pain is unbearable morphia may be used, but it is a dangerous remedy in sciatica and should be withheld as long as possible. The disease is so protracted, so liable to relapse, and the patient's *morale* so undermined by the constant worry and the sleepless nights, that the danger of contracting the morphia habit is very great. On no consideration should the patient be permitted to use the hypodermic needle himself. It is remarkable how promptly, in some cases, the injection of distilled water into the nerve will relieve the pain. Acupuncture may also be tried; the needles should be thrust deeply into the most painful spot for a distance of about 2 inches, and left for from fifteen to twenty minutes. The injection of chloroform into the nerve has also been recommended.

Electricity is an uncertain remedy. Sometimes it gives prompt relief; in other cases it may be used for weeks without the slightest benefit. It is most serviceable in the chronic cases in which there is wasting of the legs, and should be combined with massage. The galvanic current should be used; a flat electrode should be placed over the sciatic notch, and a smaller one used along the course of the nerve and its branches. In very obstinate cases nerve-stretching may be employed. It is sometimes successful; but in other instances the condition recurs and is as bad as ever.

VII. GENERAL AND FUNCTIONAL DISEASES.

I. ACUTE DELIRIUM (*Bell's Mania*).

Definition.—Acute delirium running a rapidly fatal course, with slight fever, and in which post mortem no lesions are found sufficient to account for the disease.

Cases are reported by many old writers under the term brain fever or phrenitis. Bell, at the time Superintendent of the McLean Asylum, described it * accurately under the designation, "a form of disease resembling some advanced stages of mania and fever."

The disease may set in abruptly or be preceded by a period of irritability, restlessness, and insomnia. The mental symptoms develop with rapidity and may quickly reach a grade of the most intense frenzy. There are the wildest hallucinations and outbreaks of great violence. The patient talks incessantly, but incoherently and unintelligibly. No sleep is obtained, and at last, worn out with the intensity of the muscular movements, the patient becomes utterly prostrated and assumes the sitting or recumbent posture. There may sometimes be definite salaam movements, and in a case which I saw at Westphal's clinic the patient incessantly made motions as if working a pump handle. After a period of intense bodily excitement, lasting for from twenty-four to thirty-six hours or longer, the patient can be examined, and presents the conditions which Bell described as typho-mania. The temperature ranges from 102° to 104°, or even higher. The tongue is dry, the pulse rapid and feeble; sometimes there are seen on the skin bullæ and pustules, and frequently sores from abrasion and self-inflicted injuries. Toward the close or, according to Spitzka, even during the development of the disease there may be lucid intervals. There may be petechiæ on the skin, and often there is marked congestion of the face and extremities. The duration of the disease is variable. Very acute cases may terminate within a week; others persist for two or even three weeks. The course of the disease is almost uniformly fatal. The anatomical condition is practically negative, or at any rate presents nothing distinctive. There is great venous engorgement of the vessels of the meninges and of the gray cortex. In two cases in which I made a careful microscopical examination of the gray matter there were perivascular exudation and leucocytes in the lymph sheaths and perigangliar spaces. In the inspection of fatal cases of acute delirium careful examination should be made of the lungs and ileum. It should be borne in mind that in a majority of the cases dying in this manner, there is engorgement of the bases of the lungs or even deglutition pneumonia.

The nature of the disease is quite unknown. Some of the cases suggest acute infection. Spitzka thinks that it is due to an autochthonous nerve poison.

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* American Journal of Insanity, 1849.

Diagnosis.—There are several diseases which may present identical symptoms. As Bell remarks in his paper, the first glance in many cases suggests typhoid fever, particularly when the patient is seen after the violence of the mania has subsided. He gives two instances of this which were admitted from a general hospital. Enlargement of the spleen, the occurrence of spots, and the history give clews for the separation of the cases; but there are instances in which it is at first impossible to decide. Moreover, typhoid fever may set in with the most intense delirium. The existence of fever is the most deceptive symptom, and its combination with delirium and dry tongue so commonly means typhoid fever that it is very difficult to avoid error.

Acute pneumonia may come on with violent maniacal delirium and the pulmonary symptoms may be entirely masked.

Occasionally acute uræmia sets in suddenly with intense mania, and finally subsides into a fatal coma. The condition of the urine and the absence of fever would be important diagnostic features.

The character of the delirium is quite different from that of *mania a potu*. It may be extremely difficult to differentiate acute delirium from certain cases of cortical meningitis occurring in connection with pneumonia, ulcerative endocarditis or tuberculosis, or due to extension from disease of the ear. This sets in more frequently with a chill, and there may be convulsions.

Treatment.—Even though bodily prostration is apt to come on early and be profound, in the case of a robust man free venesection might be tried. I have been criticised for this advice, but repeat it. It is not at all improbable that some of the many cases of mania in which Benjamin Rush let blood with such benefit belonged to this class of affections. Considering its remarkable calming influence in febrile delirium, the cold bath or the cold pack should be employed. Morphia and chloroform may be administered and hyoscine and the bromides may be tried. Krafft-Ebing states that Solivetti has obtained good results by the use of ergotin. Unfortunately, as asylum reports show, the disease is almost uniformly fatal.

II. PARALYSIS AGITANS

(*Parkinson's Disease; Shaking Palsy*).

Definition.—A chronic affection of the nervous system, characterized by muscular weakness, tremors, and rigidity.

Etiology.—Men are more frequently affected than women. It rarely occurs under forty, but instances have been reported in which the disease began about the twentieth year. It is by no means an uncommon affection. Direct heredity is rare, but the patients often belong to families in which there are other nervous affections. Among exciting causes may be mentioned exposure to cold and wet, and business worries and anxieties. In some instances the disease has followed directly upon severe mental shock or trauma. Cases have been described after the specific fevers. Malaria

is believed by some to be an important factor, but of this there is no satisfactory evidence.

Morbid Anatomy.—No constant lesions have been found. The similarity between certain of the features of Parkinson's disease and those of old age suggest that the affection may depend upon a premature senility of certain regions of the brain. Our organs do not age uniformly, but in some, owing to hereditary disposition, the process may be more rapid than in others. "Parkinson's disease has no characteristic lesions, but on the other hand it is not a neurosis. It has for an anatomical basis the lesions of cerebro-spinal senility, and which only differ from those of true senility in their early onset and greater intensity" (Dubief). The important changes are doubtless in the cerebral cortex.

Symptoms.—The disease begins gradually, usually in one or other hand, and the tremor may be either constant or intermittent. With this may be associated weakness or stiffness. At first these symptoms may be present only after exertion. Although the onset is slow and gradual in nearly all cases, there are instances in which it sets in abruptly after fright or trauma. When well established the disease is very characteristic, and the diagnosis can be made at a glance. The four prominent symptoms are tremor, weakness, rigidity, and the attitude.

Tremor.—This may be in the four extremities or confined to hands or feet; the head is not so commonly affected. The tremor is usually marked in the hands, and the thumb and forefinger display the motion made in the act of rolling a pill. At the wrist there are movements of pronation and supination, and, though less marked, of flexion and extension. The upper-arm muscles are rarely involved. In the legs the movement is most evident at the ankle-joint, and less in the toes than in the fingers. Shaking of the head is less frequent, but does occur, and is usually vertical, not rotatory. The rate of oscillation is about five per second. Any emotion exaggerates the movement. The attempt at a voluntary movement may check the tremor (the patient may be able to thread a needle), but it returns with increased intensity. The tremors cease, as a rule, during sleep, but persist when the muscles are at repose. The writing of the patient is tremulous and zigzag.

Weakness.—Loss of power is present in all cases, and may occur even before the tremor, but is not very striking, as tested by the dynamometer, until the late stages. The weakness is greatest where the tremor is most developed. The movements, too, are remarkably slow. There is rarely complete loss of power.

Rigidity may early be expressed in a slowness and stiffness in the voluntary movements, which are performed with some effort and difficulty, and all the actions of the patient are deliberate. This rigidity is in all the muscles, and leads ultimately to the characteristic

Attitude and Gait.—The head is bent forward, the back is bowed, and the arms are held away from the body and are somewhat flexed at the elbow-joints. The face is expressionless, and the movements of the lips are slow. The eyebrows are elevated, and the whole expression is immobile or mask-like, the so-called Parkinson's mask. The voice, as pointed out

by Buzzard, is apt to be shrill and piping, and there is often a hesitancy in beginning a sentence; then the words are uttered with rapidity, as if the patient was in a hurry. This is sometimes in striking contrast to the scanning speech of insular sclerosis. The fingers are flexed and in the position assumed when the hand is at rest; in the late stages they cannot be extended. Occasionally there is overextension of the terminal phalanges. The hand is usually turned toward the ulnar side and the attitude somewhat resembles that of advanced cases of rheumatoid arthritis. In the late stages there are contractures at the elbows, knees, and ankles. The movements of the patient are characterized by great deliberation. He rises from the chair slowly in the stooping attitude, with the head projecting forward. In attempting to walk the steps are short and hurried, and, as Trousseau remarks, he appears to be running after his centre of gravity. This is termed festination or propulsion, in contradistinction to a peculiar gait observed when the patient is pulled backward, when he makes a number of steps and would fall over if not prevented—retropulsion.

The reflexes are normal in most cases, but in a few they are exaggerated.

Of sensory disturbances Charcot has noted abnormal alterations in the temperature sense. The patient may complain of subjective sensations of heat, either general or local—a phenomenon which may be present on one side only and associated with an actual increase of the surface temperature, as much as 6° F. (Gowers). In other instances, patients complain of cold. Localized sweating may be present. The mental condition rarely shows any change.

Variations in the Symptoms.—The tremor may be absent, but the rigidity, weakness, and attitude are sufficient to make the diagnosis. The disease may be hemiplegic in character, involving only one side or even one limb. Usually these are but stages of the disease.

Diagnosis.—In well-developed cases the disease is recognized at a glance. The attitude, gait, stiffness, and mask-like expression are points of as much importance as the oscillations, and usually serve to separate the cases from senile and other forms of tremor. Disseminated sclerosis develops earlier, and is characterized by the nystagmus, and the scanning speech, and does not present the *attitude* so constant in paralysis agitans. Yet Schultze and Sachs have reported cases in which the signs of multiple sclerosis have been associated with those of paralysis. The hemiplegic form might be confounded with post-hemiplegic tremor, but the history, the mode of onset, and the greatly increased reflexes would be sufficient to distinguish the two. The Parkinsonian face is of great importance in the diagnosis of the obscure and anomalous forms.

The disease is incurable. Periods of improvement may occur, but the tendency is for the affection to proceed progressively downward. It is a slow, degenerative process and the cases last for years.

Treatment.—There is no method which can be recommended as satisfactory in any respect. Arsenic, opium, and hyoscyamine may be tried, but the friends of the patient should be told frankly that the disease is incurable, and that nothing can be done except to attend to the physical comforts of the patient.

Regulated and systematized exercises should be carried out, as in some instances ~~the~~ remarkable improvement has followed these measures

Of 1,000 cases at Allen Starr's clinic - 21 had a rheumatic history.

Other Forms of Tremor.

(*a*) *Simple Tremor.*—This is occasionally found in persons in whom it is impossible to assign any cause. It may be transient or persist for an indefinite time. It is often extremely slight, and is aggravated by all causes which lower the vitality.

(*b*) *Hereditary Tremor.*—C. L. Dana has reported remarkable cases of hereditary tremor. It occurred in all the members of one family, and beginning in infancy continued without producing any serious changes.

(*c*) *Senile Tremor.*—With advancing age tremulousness during muscular movements is extremely common, but is rarely seen under seventy. It is always a fine tremor, which begins in the hands and often extends to the muscles of the neck, causing slight movement of the head.

(*d*) *Toxic tremor* is seen chiefly as an effect of tobacco, alcohol, lead, or mercury; more rarely in arsenical or opium poisoning. In elderly men who smoke much it may be entirely due to the tobacco. One of the commonest forms of this is the alcoholic tremor, which occurs only on movement and has considerable range. Lead tremor is considered under lead poisoning, of which it constitutes a very important symptom.

(*e*) *Hysterical tremor,* which usually occurs under circumstances which make the diagnosis easy, will be considered in the section on hysteria.

III. ACUTE CHOREA

(*Sydenham's Chorea; St. Vitus's Dance*).

Definition.—A disease chiefly affecting children, characterized by irregular, involuntary contraction of the muscles, a variable amount of psychical disturbance, and a remarkable liability to acute endocarditis.

We shall speak here only of Sydenham's chorea. Senile chorea, chronic chorea, the prehemiplegic and post hemiplegic forms, and rhythmic chorea are totally different affections.

Etiology.—*Sex.*—Of 554 cases which I have analyzed from the Philadelphia Infirmary for Diseases of the Nervous System, 71 per cent were in females and 29 per cent in males. After puberty the percentage in females increases.

Age.—The disease is most common between the ages of five and fifteen. Of 522 cases, 380 occurred in this period. It is more common in the lower classes, and is rare among the negroes and native races of this continent. Morris J. Lewis has shown that the cases are most numerous when the mean relative humidity and barometric pressure are low.

Rheumatism.—A causal relationship between rheumatism and chorea has been claimed by many since the time of Bright. The English and French writers maintain the closeness of this connection; on the other hand, German authors, as a rule, regard the connection as by no means very close. Of 554 cases which I have analyzed, in 15.5 per cent there was a history of rheumatism in the family. In 88 cases, 15.8 per cent, there was a history of articular swelling, acute or subacute. In 33 cases there

were pains, sometimes described as rheumatic, in various parts, but not associated with joint trouble. If we regard all such cases as rheumatic and add them to those with manifest articular trouble, the percentage is raised to nearly 21.

We find two groups of cases in which acute arthritis is present in chorea. In one, the arthritis antedates by some months or years the onset of the chorea, and does not recur before or during the attack. In the other group, the chorea sets in with or follows immediately upon the acute arthritis. In some instances it is impossible to decide whether the joint symptoms or the movements have appeared first. It is difficult to differentiate the cases of irregular pains without definite joint affection. It is probable that many of them are rheumatic, and yet I think it would be a mistake to regard as such all cases in children in which there are complaints of vague pains in the bones or muscles—so-called growing pains. It should never be forgotten, however, that a slight articular swelling may be the sole manifestation of rheumatism in a child—so slight, indeed, that the disease may be entirely overlooked.

Heart-disease.—Endocarditis is believed by some writers to be the cause of the disease. The particles of fibrin and vegetations from the valves pass as emboli to the cerebral vessels. On this view, which we shall discuss later, chorea is the result of an embolic process occurring in the course of a rheumatic endocarditis.

Infectious Diseases.—Scarlet fever with arthritic manifestations may be a direct antecedent. Sturges states that a history of previous whooping-cough occurs more frequently in choreic than in other children, but I find no evidence of this in the Infirmary records. With the exception of rheumatic fever, there is no intimate relationship between chorea and the acute diseases incident to childhood. It may be noted in contrast to this that the so-called canine chorea is a common sequel of distemper. Chorea has been known to develop in the course of an acute pyæmia, and to follow gonorrhœa and puerperal fever.

Anæmia is less often an antecedent than a sequence of chorea, and though cases develop in children who are anæmic and in poor health, this is by no means the rule. Chorea may develop in chlorotic girls at puberty.

Pregnancy.—A choreic patient may become pregnant; more frequently the disease occurs during pregnancy; sometimes it develops post partum. Buist, of Dundee (Trans. Edin. Obs. Soc., 1895), has tabulated carefully the recorded cases to that date. Of 226 cases, in 6 the chorea preceded the pregnancy; in 105 it occurred during the pregnancy; in 31 in recurrent pregnancies; 45 cases terminated fatally, and in 16 cases the chorea developed post partum. The alleged frequency in illegitimate primiparæ is not borne out by his figures. Beginning in the first three months were 108 cases, in the second three months 70 cases, in the last three months 25 cases. The disease is often severe, and maniacal symptoms may develop.

A tendency to the disease is found in certain families. In 80 cases there was a history of attacks of chorea in other members. In one instance both mother and grandmother had been affected. High-strung, excitable,

and there are on record seven cases of embolism of the central artery of the retina (H. M. Thomas, 1901). Based upon the presence of emboli, ----

nervous children are especially liable to the disease. *Fright* is considered a frequent cause, but in a large majority of the cases no close connection exists between the fright and the onset of the disease. Occasionally the attack sets in at once. Mental worry, trouble, a sudden grief, or a scolding may apparently be the exciting cause. The strain of *education*, particularly in girls during the third hemidecade, is a most important factor in the etiology of the disease. Bright, intelligent, active-minded girls from ten to fourteen, ambitious to do well at school, often stimulated in their efforts by teachers and parents, form a large contingent of the cases of chorea in hospital and private practice. Sturges has called special attention to this *school-made* chorea as one serious evil in our modern method of forced education. *Imitation*, which is mentioned as an exciting cause, is extremely rare, and does not appear to have influenced the onset in a single case in the Infirmary records.

The disease may rapidly follow an injury or a slight surgical operation. Reflex irritation was believed to play an important *rôle* in the disease, particularly the presence of worms or genital irritation; but I have met with no instance in which the disease could be attributed to either of these causes. Local spasm, particularly of the face—the habit chorea of Mitchell—may be associated with irritation in the nostrils and adenoid growths in the vault of the pharynx, as pointed out by Jacobi.

It has been claimed by Stevens that *ocular defects* lie at the basis of many cases of chorea, and that with the correction of these the irregular movements disappear. The investigations of De Schweinitz show that ocular defects do not occur in greater proportion in choreic than in other children. A majority of the cases in which operation has been followed by relief have been instances of *tic*, local or general.

Morbid Anatomy and Pathology.—No constant lesions have been found in the nervous system in acute chorea. Vascular changes, such as hyaline transformation, exudation of leucocytes, minute hæmorrhages, and thrombosis of the smaller arteries, have been described.

Embolism of the smaller cerebral vessels has been found, ~~as might be expected in a disease with which endocarditis is so frequently associated, and based upon this fact.~~ Kirkes and others have supported what is known as the embolic theory of the disease. Endocarditis is by far the most frequent lesion in Sydenham's chorea. With no disease, not excepting rheumatism, is it so constantly associated. I have collected from the literature (to July, 1894) the records of 73 autopsies; there were 62 with endocarditis.* The endocarditis is usually of the simple variety, but the ulcerative form has occasionally been described.

We are still far from a solution of all the problems connected with chorea. Unfortunately, the word has been used to cover a series of totally diverse disorders of movement, so that there are still excellent observers who hold that chorea is only a symptom, and is not to be regarded as an etiological unit. The chorea of childhood, the disease which Sydenham described, presents, however, characteristics so unmistakable that it must

* Osler, Chorea and Choreiform Affections, 1894.

be regarded as a definite, substantive affection. We cannot discuss fully, but only indicate briefly, certain of the theories which have been advanced with regard to it. The most generally accepted view is that it is *a functional brain disorder* affecting the nerve-centres controlling the motor apparatus, an instability of the nerve-cells, brought about, one supposes by hyperæmia, another by anæmia, a third by psychical influences, a fourth by irritation, centric or peripheric. Of the actual nature of this derangement we know nothing, nor, indeed, whether the changes are primary and the result of a faulty action of the cortical cells or whether the impulses are secondarily disturbed in their course down the motor path. The predominance of the disease in females, and its onset at a time when the education of the brain is rapidly developing, are etiological facts which Sturges has urged in favor of the view that chorea is an expression of functional instability of the nerve-centres.

The *embolic theory* originally advanced by Kirkes has a solid basis of fact, but it is not comprehensive enough, as all of the cases cannot be brought within its limits. There are instances without endocarditis and without, so far as can be ascertained, plugging of cerebral vessels; and there are also cases with extensive endocarditis in which the histological examination of the brain, so far as embolism is concerned, was negative. In favor of the embolic view is the experimental production in animals of chorea by Rosenthal, and later by Money, by injecting fine particles into the carotids.

Lately, as indeed might be expected, chorea has been regarded as an *infectious disease.* Nothing definite has yet been determined. In favor of this view it has been urged, as it is impossible to refer the chorea to endocarditis or the endocarditis in all cases to rheumatism, that both have their origin in a common cause, some infectious agent, which is capable also, in persons predisposed, of exciting articular disease. Cases have been reported in scarlet fever with arthritic manifestations, in puerperal fever, and rheumatism, also after gonorrhœa, and such facts are suggestive at least of the association of the disease with infective processes. Possibly, as has been suggested by some writers, the paralytic conditions associated with chorea may be analogous to those which occur in typhoid and certain of the infectious diseases. On the other hand, there are conditions extremely difficult to harmonize with this view. The prominent psychical element is certainly one of the most serious objections, since there can be no doubt that ordinary chorea may rapidly follow a fright or a sudden emotion.

Symptoms.—Three groups of cases may be recognized—the mild, severe, and maniacal chorea.

Mild Chorea.—In this the affection of the muscles is slight, the speech is not seriously disturbed, and the general health not impaired. Premonitory symptoms are shown in restlessness and inability to sit still, a condition well characterized by the term "fidgets." There are emotional disturbances, such as crying spells, or sometimes night-terrors. There may be pains in the limbs and headache. Digestive disturbances and anæmia may be present. A change in the temperament is frequently noticed, and a docile, quiet child may become cross and irritable. After these symp-

In very rare instances more serious symptoms develop. A fatal arsenical neuritis followed in the case of a child, aged eight, who took seven drops of Fowler's solution three times a day, for ten days, then stopped for a week, - and then took seven drops three times a day for fourteen days (Cary & Gamble).

toms have persisted for a week or more the characteristic involuntary movements begin, and are often first noticed at the table, when the child spills a tumbler of water or upsets a plate. There may be only awkwardness or slight incoördination of voluntary movements, or constant irregular clonic spasms. The jerky, irregular character of the movements differentiates them from almost every other disorder of motion. In the mild cases only one hand, or the hand and face, are affected, and it may not spread to the other side.

In the second grade, the *severe form*, the movements become general and the patient may be unable to get about or to feed or undress herself, owing to the constant, irregular, clonic contractions of the various muscle groups. The speech is also affected, and for days the child may not be able to talk. Often with the onset of the severer symptoms there is loss of power on one side or in the limb most affected.

The third and most extreme form, the so-called maniacal chorea, or *chorea insaniens*, is truly a terrible disease, and may develop out of the ordinary form. These cases are more common in adult women and may develop during pregnancy.

Chorea begins, as a rule, in the hands and arms, then involves the face, and subsequently the legs. The movements may be confined to one side —hemichorea. The attack begins oftenest on the right side, though occasionally it is general from the outset. One arm and the opposite leg may be involved. In nearly one fourth of the cases speech is affected; this may amount only to an embarrassment or hesitancy, but in other instances it becomes an incoherent jumble. In very severe cases the child will make no attempt to speak. The inability is in articulation rather than in phonation. Paroxysms of panting and of hard expiration may occur, or odd sounds may be produced. As a rule the movements cease during sleep.

A prominent symptom is muscular weakness, usually no more than a condition of paresis. The loss of power is slight, but the weakness may be shown by an enfeebled grip or by a dragging of the leg or limping. In his original account Sydenham refers to the "unsteady movements of one of the legs, which the patient drags." There may be extreme paresis with but few movements—the paralytic chorea of Todd. Occasionally a local paralysis or weakness remains after the attack.

It is doubtful whether choreic spasms extend to the muscles of organic life. The rapid action and disturbed rhythm of the heart present nothing peculiar to the disease, and there is no support for the view that irregular contractions occur in the papillary muscles.

Heart Symptoms.—*Neurotic.*—As so many of the subjects of chorea are nervous girls, it is not surprising that a common symptom is a rapidly acting heart. Irregularity, however, is not so special a feature in chorea as rapidity. The patients seldom complain of pain about the heart.

Hæmic Murmurs.—With anæmia and debility, not uncommon associates of chorea in the third or fourth week, we find a corresponding cardiac condition. The impulse is diffuse, perhaps wavy in thin children. The carotids throb visibly, and in the recumbent posture there may be

pulsation in the cervical veins. On auscultation a systolic murmur is heard at the base, perhaps, too, at the apex, soft and blowing in quality.

Endocarditis.—As in rheumatism, so in chorea, acute valvulitis rarely gives evidence of its presence by symptoms. It must be sought, and clinical experience has shown that it is usually associated with murmurs at one or other of the cardiac orifices.

For the guidance of the practitioner the following statements may be made:

(1) In thin, nervous children a systolic murmur of soft quality is extremely common at the base, particularly at the second left costal cartilage, and is probably of no moment.

(2) A systolic murmur of maximum intensity at the apex, and heard also along the left sternal margin, is not uncommon in anæmic, enfeebled states, and does not necessarily indicate either endocarditis or insufficiency.

(3) A murmur of maximum intensity at apex, with rough quality, and transmitted to axilla or angle of scapula, indicates an organic lesion of the mitral valve, and is usually associated with signs of enlargement of the heart.

(4) When in doubt it is much safer to trust to the evidence of eye and hand than to that of the ear. If the apex beat is in the normal position, and the area of dulness not increased vertically or to the right of the sternum, there is probably no serious valvular disease.

(5) The endocarditis of chorea is almost invariably of the simple or warty form, and in itself is not dangerous; but it is apt to lead to those sclerotic changes in the valve which produce incompetency. Of 140 patients examined more than two years after the attack,* I found the heart normal in 51; in 17 there was functional disturbance, and 72 presented signs of organic heart-disease.

(6) Pericarditis is an occasional complication of chorea, usually in cases with well-marked rheumatism.

Sensory Disturbances.—Pain in the affected limbs is not common. Occasionally there is soreness on pressure. There are cases, usually of hemichorea, in which pain in the limbs is a marked symptom. Weir Mitchell has spoken of these as *painful choreas.* Tender points along the lines of emergence of the spinal nerves or along the course of the nerves of the limbs are rare.

Psychical disturbances are common, though in a majority of the cases slight in degree. Irritability of temper, marked wilfulness, and emotional outbreaks may indicate a complete change in the character of the child. There is deficiency in the powers of concentration, the memory is enfeebled, and the aptitude for study is lost. Rarely there is progressive impairment of the intellect with termination in actual dementia. Acute melancholia has been described (Edes). Hallucinations of sight and hearing may occur. Patients may behave in an odd and strange manner and do all sorts of meaningless acts. By far the most serious manifestation of

* Monograph on Chorea, 1894.

this character is the maniacal delirium, occasionally associated with the very severe cases—*chorea insaniens*. Usually the motor disturbance in these cases is aggravated, but it has been overlooked and patients have been sent to an asylum.

The psychical element in chorea is apt to be neglected by the practitioner. It is always a good plan to tell the parents that it is not the muscles alone of the child which are affected, but that the general irritability and change of disposition, so often found, really form part of the disease.

The condition of the *reflexes* in chorea is usually normal. Trophic lesions rarely occur in chorea unless, as some writers have done, we regard the joint troubles as arthropathies occurring in the course of a cerebrospinal disease.

Fever is not, as a rule, present in chorea unless complications exist. There may be the most intense and violent movements without any rise of temperature. I have seen instances, however, in which without apparently any visceral or articular disturbances there was slight daily fever. H. A. Hare states that in monochorea the temperature on the affected side may be elevated; but this is not an invariable rule. Fever is found with an acute arthritis, when there is marked endocarditis or pericarditis, though the former may certainly occur with little if any rise in temperature, and in the cases of maniacal chorea, in which the fever may range from 102° to 104°.

Cutaneous Affections.—The pigmentation, which is not uncommon, is due to the arsenic. Herpes zoster occasionally occurs. Certain skin eruptions, usually regarded as rheumatic in character, are not uncommon. Erythema nodosum has been described and I have seen several cases with a purpuric urticaria. There may, indeed, be the more aggravated condition of rheumatic purpura, known as Schönlein's *peliosis rheumatica*. Subcutaneous fibrous nodules, which have been noted by English observers in many cases of chorea, associated with rheumatism, are extremely rare in this country.

Duration and Termination.—From eight to ten weeks is the average duration of an attack of moderate severity. Chronic chorea rarely follows the minor disease which we have been considering. The cases described under this designation in children are usually instances of cerebral sclerosis or Friedreich's ataxia; but occasionally an attack which has come on in the ordinary way persists for months or years, and recovery ultimately takes place. A slight grade of chorea, particularly noticeable under excitement, may persist for months in nervous children.

The tendency of chorea to recur has been noticed by all writers since Sydenham first made the observation. Of 410 cases analyzed for this purpose, 240 had one attack, 110 had two attacks, 35 three attacks, 10 four attacks, 12 five attacks, and 3 six attacks. The recurrence is apt to be vernal.

Recovery is the rule in children. The statistics of out-patients' departments are not favorable for determining the mortality. A reliable estimate is that of the Collective Investigation Committee of the British Medi-

cal Association, in which 9 deaths were reported among 439 cases, about 2 per cent.

The paralysis rarely persists. Mental dulness may be present for a time, but usually passes away; permanent impairment of the mind is an exceptional sequence.

Diagnosis.—There are few diseases which present more characteristic features, and in a majority of instances the nature of the trouble is recognized at a glance; but there are several affections in children which may simulate and be mistaken for it.

(*a*) Multiple and diffuse cerebral sclerosis. The cases are often mistaken for ordinary chorea, and have been described in the literature as *chorea spastica.*

There are doubtless chronic changes in the cortex. As a rule, the movements are readily distinguishable from those of true chorea, but the simulation is sometimes very close; the onset in infancy, the impaired intelligence, increased reflexes and in some instances rigidity, and the chronic course of the disease, separate them sharply from true chorea.

(*b*) Friedreich's ataxia. Cases of this well-characterized disease were formerly classed as chorea. The slow, irregular, incoördinate movements, the scoliosis, the scanning speech, the early talipes, the nystagmus, and the family character of the disease are points which should render the diagnosis easy.

(*c*) In rare cases the paralytic form of chorea may be mistaken for polio-myelitis or, when both legs are affected, for paraplegia of spinal origin; but this can only be the case when the choreic movements are very slight.

(*d*) Hysteria may simulate chorea minor most closely, and unless there are other manifestations it may be impossible to make a diagnosis. Most commonly, however, the movements in the so-called hysterical chorea are rhythmic and differ entirely from those of ordinary chorea.

(*e*) As mentioned above, the mental symptoms in maniacal chorea may mask the true nature of the disease and patients have even been sent to the asylum.

Treatment.—Abnormally bright, active-minded children belonging to families with pronounced neurotic taint should be carefully watched from the ages of eight to fifteen and not allowed to overtax their mental powers. So frequently in children of this class does the attack of chorea date from the worry and stress incident to school examinations that the competition for prizes or places should be emphatically forbidden.

The treatment of the attack consists largely in attention to hygienic measures, with which alone, in time, a majority of the cases recover. Parents should be told to scan gently the faults and waywardness of choreic children. The psychical element, strongly developed in so many cases, is best treated by quiet and seclusion. The child should be confined to bed in the recumbent posture, and mental as well as bodily quiet enjoined. In private practice this is often impossible, but with well-to-do patients the disease is always serious enough to demand the assistance of a skilled nurse. Toys and dolls should not be allowed at first, for the child should

be kept amused without excitement. The rest allays the hyper-excitability and reduces to a minimum the possibility of damage to the valve segments should endocarditis exist. Time and again have I seen very severe cases which had resisted treatment for weeks outside a hospital become quiet and the movements subside after two or three days of absolute rest in bed.

The child should be kept apart from other children and, if possible, from other members of the family, and should see only those persons directly concerned with the nursing of the case. Though irksome and troublesome to carry out, this is an important part of the treatment. In the latter period of the disease daily rubbings may be resorted to with great benefit.

The medical treatment of the disease is unsatisfactory; with the exception of arsenic, no remedy seems to have any influence in controlling the progress of the affection. Without any specific action, it certainly does good in many cases, probably by improving the general nutrition. It is conveniently given in the form of Fowler's solution, and the good effects are rarely seen until maximum doses are taken. It may be given as Martin originally advised (1813); he began "with five drops and increased one drop every day, until it might begin to disagree with the stomach or bowels." When the dose of 15 minims is reached, it may be continued for a week, and then again increased, if necessary, every day or two, until physiological effects are manifest. On the occurrence of these the drug should be stopped for three or four days. The practice of resuming the administration with smaller doses is rarely necessary, as tolerance is usually established and we can begin with the dose which the child was taking when the symptoms of saturation occurred. I have frequently given as much as 25 minims three times a day. Usually the signs of saturation are trivial but plain, and I have never seen any ill effects from the large doses, although I have heard recently of a case of arsenical neuritis due to the administration of Fowler's solution in chorea.

Of other medicines, strychnine, the zinc compounds, nitrate of silver, bromide of potassium, belladonna, chloral, and especially cimicifuga, have been recommended, and may be tried in obstinate cases.

For its tonic effect electricity is sometimes useful; but it is not necessary as a routine treatment. The question of gymnastics is an important one. Early in the disease, when the movements are active, they are not advisable; but during convalescence carefully graduated exercises are undoubtedly beneficial. It is not well, however, to send a choreic child to a school gymnasium, as the stimulus of the other children and the excitement of the romping, violent play is very prejudicial.

Other points in treatment may be mentioned. It is important to regulate the bowels and to attend carefully to the digestive functions. For the anæmia so often present preparations of iron are indicated.

In the severe cases with incessant movements, sleeplessness, dry tongue, and delirium, the important indication is to procure rest, for which purpose chloral may be freely given, and, if necessary, morphia. Chloroform inhalations may be necessary to control the intensity of the paroxysms,

but the high rate of mortality in this class of cases illustrates how often our best endeavors are fruitless. The wet pack is sometimes very soothing and should be tried. As these patients are apt to sink rapidly into a low typhoid state with heart weakness, a supporting treatment is required from the outset.

Cases are found now and then which drag on from month to month without getting either better or worse and resist all modes of treatment. Change of air and scene is sometimes followed by rapid improvement, and in these cases the treatment by rest and seclusion should always be given a full trial.

In all cases care should be taken to examine the nostrils, and glaring ocular defects should be properly corrected either by glasses or, if necessary, by operation.

After the child has recovered from the attack, the parents should be warned that return of the disease is by no means infrequent, and is particularly liable to follow overwork at school or debilitating influences of any kind. These relapses are apt to occur in the spring. Sydenham advised purging in order to prevent the vernal recurrence of the disease.

IV. OTHER AFFECTIONS DESCRIBED AS CHOREA.

(*a*) **Chorea Major; Pandemic Chorea.**—The common name, St. Vitus's dance, applied to chorea has come to us from the middle ages, when under the influence of religious fervor there were epidemics characterized by great excitement, gesticulations, and dancing. For the relief of these symptoms, when excessive, pilgrimages were made, and in the Rhenish provinces, particularly to the Chapel of St. Vitus in Zebern. Epidemics of this sort have occurred also during this century, and descriptions of them among the early settlers in Kentucky have been given by Robertson and Yandell. It was unfortunate that Sydenham applied the term chorea to an affection in children totally distinct from this chorea major, which is in reality an hysterical manifestation under the influence of religious excitement.

(*b*) **Habit Spasm (Habit Chorea); Convulsive Tic** (of the French).

Two groups of cases may be recognized under the designation of habit spasm—one in which there are simply localized spasmodic movements, and the other in which, in addition to this, there are explosive utterances and psychical symptoms, a condition to which French writers have given the name *tic convulsif*.

(1) *Habit Spasm.*—This is found chiefly in childhood, most frequently in girls from seven to fourteen years of age (Mitchell). In its simplest form there is a sudden, quick contraction of certain of the facial muscles, such as rapid winking or drawing of the mouth to one side, or the neck muscles are involved and there are unilateral movements of the head. The head is given a sudden, quick shake, and at the same time the eyes wink. A not infrequent form is the shrugging of one shoulder. The grimace or movement is repeated at irregular intervals, and is much aggravated by emotion. A short inspiratory sniff is not an uncommon symp-

Impulsive tic.

Head-shaking in infants (see Thomsen's article in Jacobi Festschrift).

tom. The cases are found most frequently in children who are "out of sorts," or who have been growing rapidly, or who have inherited a tendency to neurotic disorders. Allied to or associated with this are some of the curious tricks of children. A boy at my clinic was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the forefinger. Hartley Coleridge is said to have had a somewhat similar trick, only he bit his arm. In all these cases the habits of the child should be examined carefully, the nose and vault of the pharynx thoroughly inspected, and the eyes accurately tested. As a rule the condition is transient, and after persisting for a few months or longer gradually disappears. Occasionally a local spasm persists —twitching of the eyelids, or the facial grimace.

(2) *Tic Convulsif* (*Gilles de la Tourette's Disease*).—This remarkable affection, often mistaken for chorea, more frequently for habit spasm, is really a psychosis allied to hysteria, though in certain of its aspects it has the features of monomania. The disease begins, as a rule, in young children, occurring as early as the sixth year, though it may develop after puberty. There is usually a markedly neurotic family history. The special features of the complaint are:

(*a*) Involuntary muscular movements, usually affecting the facial or brachial muscles, but in aggravated cases all the muscles of the body may be involved and the movements may be extremely irregular and violent.

(*b*) Explosive utterances, which may resemble a bark or an inarticulate cry. A word heard may be mimicked at once and repeated over and over again, usually with the involuntary movements. To this the term *echolalia* has been applied. A much more distressing disturbance in these cases is *coprolalia*, or the use of bad language. A child of eight or ten may shock its mother and friends by constantly using the word *damn* when making the involuntary movements, or by uttering all sorts of obscene words. Occasionally actions are mimicked—*echokinesis*.

(*c*) Associated with some of these cases are curious mental disturbances; the patient becomes the subject of a form of obsession or a fixed idea. In other cases the fixed idea takes the form of the impulse to touch objects, or it is a fixed idea about words—onomatomania—or the patient may feel compelled to count a number of times before doing certain actions—arithmomania.

The disease is well marked and readily distinguished from ordinary chorea. The movements have a larger range and are explosive in character. Tourette regards the coprolalia as the most distinctive feature of the disease. The prognosis is doubtful. I have, however, known recovery to follow.

(*c*) **Saltatory Spasm** (*Latah; Myriachit; Jumpers*).—Bamberger has described a disease in which when the patient attempted to stand there were strong contractions in the leg muscles, which caused a jumping or springing motion. This occurs only when the patient attempts to stand. The affection has occurred in both men and women, more frequently in the former, and the subjects have usually shown marked neurotic tendencies. In many cases the condition has been transitory; in others it has persisted

for years. Remarkable affections similar to this in certain points occur as a sort of endemic neurosis. One of the most striking of these occurs among the "jumping Frenchmen" of Maine and Canada. As described by Beard and Thornton, the subjects are liable on any sudden emotion to jump violently and utter a loud cry or sound, and will obey any command or imitate any action without regard to its nature. The condition of echolalia is present in a marked degree. The "jumping" prevails in certain families.

A very similar disease prevails in parts of Russia and in Java, where it is known by the names of myriachit and latah, the chief feature of which is mimicry by the patient of everything he sees or hears.

(*d*) **Chronic Chorea** (*Huntington's Chorea*).—An affection characterized by irregular movements, disturbance of speech, and gradual dementia. It is frequently hereditary. The disease has no connection with Sydenham's chorea, and it is unfortunate that the term was applied to it. It was described by Huntington, of Pomeroy, Ohio, at the time a practitioner on Long Island, and he gave in three brief paragraphs the salient points in connection with the disease—namely, the hereditary nature, the association with psychical troubles, and the late onset—between the thirtieth and fortieth years. The disease seems common in this country, and many cases have been reported by Clarence King, Sinkler, and others. I have seen it in two Maryland families within the past few years. Under the term chronic chorea may be grouped the hereditary form and the cases which come on without family disposition, either at middle life or, more commonly, in the aged—senile chorea. It is doubtful whether the cases in children with chronic choreiform movements, often with mental weakness and spastic condition of the legs, should go into this category.

The hereditary character of the disease is very striking; it has been traced through four or five generations. Huntington's father and grandfather, also physicians, had treated the disease in the family which he described. Osborn, of East Hampton, L. I., writes (Jan. 28th, 1898) that the disease still continues to recur in certain families described by Huntington, as it has done, so it is said, for fully two centuries. An identical affection occurs without any hereditary disposition. The age of onset is late, rarely before the thirtieth or the thirty-fifth year.

The symptoms are very characteristic. The irregular movements are usually first seen in the hands, and the patient has slight difficulty in performing delicate manipulations or in writing. When well established the movements are disorderly, irregular, incoördinate rather than choreic, and have not the sharp, brusque motion of Sydenham's chorea. In the face there are slow, involuntary grimaces. In a well-developed case the gait is irregular, swaying, and somewhat like that of a drunken man. The speech is slow and difficult, the syllables are badly pronounced and indistinct, but not definitely staccato. The mental impairment leads finally to dementia.

Very few post mortems have been made. No characteristic lesions have been found. Atrophy of the convolutions, chronic meningo-encephalitis, and vascular changes have usually been present, the conditions which one

would expect to find in chronic dementia. The recent study of two cases by Facklan (Arch. f. Psychiatrie, 30) confirms the view expressed in former editions that the disease is a chronic meningo-encephalitis with atrophy of the convolutions. The cord and peripheral nerves he found perfectly healthy. The affection is evidently a neuro-degenerative disorder, and has no connection with the simple chorea of childhood.

(*e*) **Rhythmic or Hysterical Chorea.**—This is readily recognized by the rhythmical character of the movements. It may affect the muscles of the abdomen, producing the salaam convulsion, or involve the sterno-mastoid, producing a rhythmical movement of the head, or the psoas, or any group of muscles. In its orderly rhythm it resembles the canine chorea.

V. INFANTILE CONVULSIONS (*Eclampsia*).

Convulsive seizures similar to those of epilepsy are not infrequent in children and in adults. The fit may indeed be identical with epilepsy, from which the condition differs in that when the cause is removed there is no tendency for the fits to recur. Occasionally, however, the convulsions in children continue and develop into true epilepsy.

Etiology.—A convulsion in a child may be due to many causes, all of which lead to an unstable condition of the nerve-centres, permitting of sudden, excessive, and temporary nervous discharges. The following are the most important of them:

(1) Debility, resulting usually from gastro-intestinal disturbance. Convulsions frequently supervene toward the close of an attack of entero-colitis and recur, sometimes proving fatal. Morris J. Lewis has shown that the death-rate in children from eclampsia rises steadily with that of gastro-intestinal disorders.

(2) Peripheral irritation. Dentition alone is rarely a cause of convulsions, but is often one of several factors in a feeble, unhealthy infant. The greatest mortality from convulsions is during the first six months, before the teeth have really cut through the gums. Other irritative causes are the overloading of the stomach with indigestible food. It has been suggested that some of these cases are toxic, owing to the absorption of poisonous ptomaines. Worms, to which convulsions are so frequently attributed, probably have little influence. Among other sources possible are phimosis and otitis.

(3) Rickets. The observation of Sir William Jenner upon the association of rickets and convulsions has been amply confirmed. The spasms may be laryngeal, the so-called child-crowing, which, though convulsive in nature, can scarcely be reckoned under eclampsia. The influence of this condition is more apparent in Europe than in this country, although rickets is a common disease, particularly among the colored people. Spasms, local or general, in rickets are probably associated with the condition of debility and malnutrition and with cranio-tabes.

(4) Fever. In young children the onset of the infectious diseases is frequently with convulsions, which often take the place of a chill in the adult.

It is not known upon what they depend. Scarlet fever, measles, and pneumonia are most often preceded by convulsions.

(5) Congestion of the brain. That extreme engorgement of the blood-vessels may produce convulsions is shown by their occasional occurrence in severe whooping-cough, but their rarity in this disease really indicates how small a part mechanical congestion plays in the production of fits.

(6) Severe convulsions usher in or accompany many of the serious diseases of the nervous system in children. In more than 50 per cent of the cases of infantile hemiplegia the affection follows severe convulsions. They less frequently precede a spinal paralysis. They occur with meningitis, tuberculous or simple, and with tumors and other lesions of the brain.

And, lastly, convulsions may occur immediately after birth and persist for weeks or months. In such instances there has probably been meningeal hæmorrhage or serious injury to the cortex.

The most important question is the relation of convulsions in children to true epilepsy. In Gowers' figures of 1,450 cases of epilepsy, the attacks began in 180 during the first three years of life. Of 460 cases of epilepsy in children which I have analyzed, in 187 the fits began within the first three years. Of the total list the greatest number, 74, was in the first year. In nearly all these instances there was no interruption in the convulsions. That convulsions in early infancy are necessarily followed by epilepsy in after life is certainly a mistake.

Symptoms.—The attack may come on suddenly without any warning; more commonly it is preceded by a stage of restlessness, accompanied by twitching and perhaps grinding of the teeth. It is rarely so complete in its stages as true epilepsy. The spasm begins usually in the hands, most commonly in the right hand. The eyes are fixed and staring or are rolled up. The body becomes stiff and breathing is suspended for a moment or two by tonic spasm of the respiratory muscles, in consequence of which the face becomes congested. Clonic convulsions follow, the eyes are rolled about, the hands and arms twitch, or are flexed and extended in rhythmical movements, the face is contorted, and the head is retracted. The attack gradually subsides and the child sleeps or passes into a state of stupor. Following indigestion the attack may be single, but in rickets and intestinal disorders it is apt to be repeated. Sometimes the attacks follow each other with great rapidity, so that the child never rouses but dies in a deep coma. If the convulsion has been limited chiefly to one side there may be slight paresis after recovery, or in instances in which the convulsions usher in infantile hemiplegia, when the child arouses, one side is completely paralyzed. During the fit the temperature is often raised. Death rarely occurs from the convulsion itself, except in debilitated children or when the attacks recur with great frequency. In the so-called hydrocephaloid state in connection with protracted diarrhœa convulsions may close the scene.

Diagnosis.—Coming on when the subject is in full health, the attack is probably due either to an overloaded stomach, to some peripheral irritation, or occasionally to trauma. Setting in with high fever and vomiting, it may indicate the onset of an exanthem, or occasionally be the primary symptom of encephalitis, or whatever the condition is which causes infan-

tile hemiplegia. When the attack is associated with debility and with rickets the diagnosis is easily made. The carpopedal spasms and pseudo-paralytic rigidity which are often associated with rickets, laryngismus stridulus, and the hydrocephaloid state are usually confined to the hands and arms and are intermittent and usually tonic. The convulsions associated with tumor or which follow infantile hemiplegia are usually at first Jacksonian in character. After the second year convulsive seizures which come on irregularly without apparent cause and recur while the child is apparently in good health are likely to prove true epilepsy.

Prognosis.—Convulsions play an important part in infantile mortality. In Morris J. Lewis's table of deaths in children under ten, 8.5 per cent were ascribed to convulsions. West states that 22.35 per cent of deaths under one year are caused by convulsions, but this is too high an estimate for this country. In chronic diarrhœa convulsions are usually of ill omen. Those ushering in fevers are rarely serious, and the same may be said of the fits associated with indigestion and peripheral irritation.

Treatment.—Every source of irritation should be removed. If associated with indigestible food, a prompt emetic should be given, followed by an enema. The teeth should be examined, and if the gum is swollen, hot, and tense, it may be lanced; but never if it looks normal. When seen at first, if the paroxysm is severe, no time should be lost by giving a hot bath, but chloroform should be given at once, and repeated if necessary. A child is so readily put under chloroform and with such a small quantity that this precedure is quite harmless and saves much valuable time. The practice is almost universal of putting the child into a warm bath, and if there is fever the head may be douched with cold water. The temperature of the bath should not be above 95° or 96°. The very hot bath is not suitable, particularly if the fits are due to indigestion. After the attack an ice-cap may be placed upon the head. If there is much irritability, particularly in rickets and in severe diarrhœa, small doses of opium will be found efficacious. When the convulsions recur after the child comes from under the influence of chloroform it is best to place it rapidly under the influence of opium, which may be given as morphia hypodermically, in doses of from one twenty-fifth to one thirtieth of a grain for a child of one year. Other remedies recommended are chloral by enema, in 5-grain doses, and nitrite of amyl. After the attack has passed the bromides are useful, of which 5 to 8 grains may be given in a day to a child a year old. Recurring convulsions, particularly if they come on without special cause, should receive the most thorough and careful treatment with bromides. When associated with rickets the treatment should be directed to improving the general condition.

VI. EPILEPSY.

Definition.—An affection of the nervous system characterized by attacks of unconsciousness, with or without convulsions.

The transient loss of consciousness without convulsive seizures is known

as *petit mal;* the loss of consciousness with general convulsive seizures is known as *grand mal.* Localized convulsions, occurring usually without loss of consciousness, are known as epileptiform, or more frequently as Jacksonian or cortical epilepsy.

Etiology.—*Age.*—In a large proportion of all cases the disease begins before puberty. Of the 1,450 cases observed by Gowers, in 422 the disease began before the tenth year, and three fourths of the cases began before the twentieth year. Of 460 cases of epilepsy in children which I have analyzed the age of onset in 427 was as follows: First year, 74; second year, 62; third year, 51; fourth year, 24; fifth year, 17; sixth year, 18; seventh year, 19; eighth year, 23; ninth year, 17; tenth year, 27; eleventh year, 17; twelfth year, 18; thirteenth year, 15; fourteenth year, 21; fifteenth year, 34. Arranged in hemidecades the figures are as follows: From the first to the fifth year, 229; from the fifth to the tenth year, 104; from the tenth to the fifteenth year, 95. These figures illustrate in a striking manner the early onset of the disease in a large proportion of the cases. It is well always to be suspicious of epilepsy developing in the adult, for in a majority of such cases the convulsions are due to a local lesion.

Sex.—No special influence appears to be discoverable in this relation, certainly not in children. Of 433 cases in my tables, 232 were males and 203 were females, showing a slight predominance of the male sex. After puberty unquestionably, if a large number of cases are taken, the males are in excess. The figures of Sieveking and Reynolds show that the disease is rather more prevalent in females than in males.

Heredity.—Much stress has been laid upon this by many authors as an important predisposing cause, and the statistics collected give from 9 to over 40 per cent. Gowers gives 35 per cent for his cases, which have special value apart from other statistics embracing large numbers of epileptics in that they were collected by him in his own practice. In our figures it appears to play a minor *rôle.* In the Infirmary list there were only 31 cases in which there was a history of marked neurotic taint, and only 3 in which the mother herself had been epileptic. In the Elwyn cases, as might be expected, the percentage is larger. Of the 126 there was in 32 a family history of nervous derangement of some sort, either paralysis, epilepsy, marked hysteria, or insanity. It is interesting to note that in this group, in which the question of heredity is carefully looked into, there were only two in which the mother had had epilepsy, and not one in which the father had been affected. Indeed, I was not a little surprised to find in the list of my cases that hereditary influences played so small a part. I have heard this opinion expressed by certain French physicians, notably Marie, who in writing also upon the question takes strong grounds against heredity as an important factor in epilepsy.

While, then, it may be said that direct inheritance is comparatively uncommon, yet the children of neurotic families in which neuralgia, insanity, and hysteria prevail are more liable to fall victims to the disease.

Chronic alcoholism in the parents is regarded by many as a potent predisposing factor in the production of epilepsy. Echeverria has analyzed 572 cases bearing upon this point and divided them into three classes, of

which 257 cases could be traced directly to alcohol as a cause; 126 cases in which there were associated conditions, such as syphilis and traumatism; 189 cases in which the alcoholism was probably the result of the epilepsy. Figures equally strong are given by Martin, who found in 150 insane epileptics 83 with a marked history of parental intemperance. Of the 126 Elwyn cases, in which the family history on this point was carefully investigated, a definite statement was found in only 4 of the cases.

Syphilis.—This in the parents is probably less a predisposing than an actual cause of epilepsy, which is the direct outcome of local cerebral manifestations. There is no reason for recognizing a special form of syphilitic epilepsy. On the other hand, convulsive seizures due to acquired syphilitic disease of the brain are very common.

Alcohol.—Severe epileptic convulsions may occur in steady drinkers.

Of exciting causes fright is believed to be important, but is less so, I think, than is usually stated. Trauma is present in a certain number of instances. An important group depends upon a local disease of the brain existing from childhood, as seen in the post-hemiplegic epilepsy. Occasionally cases follow the infectious fevers. Masturbation has been stated to be a special cause, but its influence is probably overrated. A large group of convulsive seizures allied to epilepsy are due to some toxic agent, as in lead poisoning and in uræmia. Great stress was laid upon reflex causes, such as dentition and worms, the irritation of a cicatrix, some local affection, such as adherent prepuce, or a foreign body in the ear or the nose. In many of these cases the fits cease after the removal of the cause, so that there can be no question of the association between the two. In others the attacks persist. Genuine cases of reflex epilepsy are, I believe, rare. A remarkable instance of it occurred at the Philadelphia Infirmary for Diseases of the Nervous System in the case of a man with a testis in the inguinal canal, pressure upon which would cause a typical fit. Removal of the organ was followed by cure.

Epilepsy has been thought to be associated with disturbance of the heart's action, and some have spoken of a special cardiac epilepsy, particularly in cases in which there is palpitation or slowing of the action prior to the onset. Epileptic seizures may occur during the passage of a gall-stone or occasionally during the removal of pleuritic fluid. Indigestion and gastric troubles are extremely common in epilepsy, and in many instances the eating of indigestible articles seems to precipitate an attack.

An attempt to associate genuine epilepsy with eye-strain has signally failed.

Symptoms.—(1) Grand Mal.—Preceding the fits there is usually a localized sensation, known as an *aura*, in some part of the body. This may be somatic, in which the feeling comes from some particular region in the periphery, as from the finger or hand, or is a sensation felt in the stomach or about the heart. The peripheral sensations preceding the fit are of great value, particularly those in which the aura always occurs in a definite region, as in one finger or toe. It is the equivalent of the signal symptom in a fit from a brain tumor. The varieties of these sensations are numerous. The epigastric sensations are most common. In these the

patient complains of an uneasy sensation in the epigastrium or distress in the intestines, or the sensation may not be unlike that of heart-burn and may be associated with palpitation. These groups are sometimes known as pneumogastric auræ or warnings.

Of psychical auræ one of the most common, as described by Hughlings Jackson, is the vague, dreamy state, a sensation of strangeness or sometimes of terror. The auræ may be associated with special senses; of these the most common are the visual, consisting of flashes of light or sensations of color; less commonly, distinct objects are seen. The auditory auræ consist of noises in the ear, odd sounds, musical tones, or occasionally voices. Olfactory and gustatory auræ, unpleasant tastes and odors, are rare.

Occasionally the fit may be preceded not by an aura, but by certain movements; the patient may turn round rapidly or run with great speed for a few minutes, the so-called epilepsia procursiva. In one of the Elwyn cases the lad stood on his toes and twirled with extraordinary rapidity, so that his features were scarcely recognizable. At the onset of the attack the patient may give a loud scream or yell, the so-called epileptic cry. The patient drops as if shot, making no effort to guard the fall. In consequence of this epileptics frequently injure themselves, cutting the face or head or burning themselves. In the attack, as described by Hippocrates, "the patient loses his speech and chokes, and foam issues from the mouth, the teeth are fixed, the hands are contracted, the eyes distorted, he becomes insensible, and in some cases the bowels are affected. And these symptoms occur sometimes on the left side, sometimes on the right, and sometimes on both." The fit may be described in three stages:

(*a*) *Tonic Spasm.*—The head is drawn back or to the right, and the jaws are fixed. The hands are clinched and the legs extended. This tonic contraction affects the muscles of the chest, so that respiration is impeded and the initial pallor of the face changes to a dusky or livid hue. The muscles of the two sides are unequally affected, so that the head and neck are rotated or the spine is twisted. The arms are usually flexed at the elbows, the hand at the wrist, and the fingers are tightly clinched in the palm. This stage lasts only a few seconds, and then the

(*b*) *Clonic stage* begins. The muscular contractions become intermittent; at first tremulous or vibratory, they gradually become more rapid and the limbs are jerked and tossed about violently. The muscles of the face are in constant clonic spasm, the eyes roll, the eyelids are opened and closed convulsively. The movements of the muscles of the jaw are very forcible and strong, and it is at this time that the tongue is apt to be caught between the teeth and lacerated. The cyanosis, marked at the end of the tonic stage, gradually lessens. A frothy saliva, which may be blood-stained, escapes from the mouth. The fæces and urine may be discharged involuntarily. The duration of this stage is variable. It rarely lasts more than one or two minutes. The contractions become less violent and the patient gradually sinks into the condition of

(*c*) *Coma.* The breathing is noisy or even stertorous, the face congested, but no longer intensely cyanotic. The limbs are relaxed and the

unconsciousness is profound. After a variable time the patient can be aroused, but if left alone he sleeps for some hours and then awakes, complaining only of slight headache or mental confusion.

In some cases one attack follows the other with great rapidity and consciousness is not regained. This is termed the *status epilepticus*, an exceptional condition, in which the patient may die of exhaustion, consequent upon the repeated attacks. In it the temperature is usually elevated.

After the attack the reflexes are sometimes absent; more frequently they are increased and the ankle clonus can usually be obtained. The state of the urine is variable, particularly as regards the solids. The quantity is usually increased after the attack, and albumin is not infrequently present.

Post-epileptic symptoms are of great importance. The patient may be in a trance-like condition, in which he performs actions of which subsequently he has no recollection. More serious are the attacks of mania, in which the patient is often dangerous and sometimes homicidal. It is held by good authorities that an outbreak of mania may be substituted for the fit. And, lastly, the mental condition of an epileptic patient is often seriously impaired, and profound defects are common.

Paralysis, which rarely follows the epileptic fit, is usually hemiplegic and transient.

Slight disturbances of speech also may occur; in some instances forms of sensory aphasia.

The attacks may occur at night, and a person may be epileptic for years without knowing it. As Trousseau truly remarks, when a person tells us that in the night he has incontinence of urine and awakes in the morning with headache and mental confusion, and complains of difficulty in speech owing to the fact that he has bitten his tongue; if, also, there are on the skin of the face and neck purpuric spots, the probability is very strong indeed that he is subject to nocturnal epilepsy.

(2) **Petit Mal.**—This is epilepsy without the convulsions. The attack consists of transient unconsciousness, which may come on at any time, accompanied or unaccompanied by a feeling of faintness and vertigo. Suddenly, for example, at the dinner table, the subject stops talking and eating, the eyes become fixed, and the face slightly pale. Anything which may have been in the hand is usually dropped. In a moment or two consciousness is regained and the patient resumes conversation as if nothing had happened. In other instances there is slight incoherency or the patient performs some almost automatic action. He may begin to undress himself and on returning to consciousness find that he has partially disrobed. He may rub his beard or face, or may spit about in a careless way. In other attacks the patient may fall without convulsive seizures. A definite aura is rare. Though transient, unconsciousness and giddiness are the most constant manifestations of *petit mal;* there are many other equivalent manifestations, such as sudden jerkings in the limbs, sudden tremor, or a sudden visual sensation. Gowers mentions no less than seventeen different manifestations of *petit mal.* Occasionally there are cases in which the patient

has a sensation of losing his breath and may even get red in the face. I have seen such attacks also in children.

After the attack the patient may be dazed for a few seconds and perform certain automatic actions, which may seem to be volitional. As mentioned, undressing is a common action, but all sorts of odd actions may be performed, some of which are awkward or even serious. One of my patients after an attack was in the habit of tearing anything he could lay hands on, particularly books. Violent actions have been committed and assaults made, frequently giving rise to questions which come before the courts. This condition has been termed masked epilepsy, or *epilepsia larvata*.

In a majority of the cases of *petit mal* convulsions finally occur, at first slight, but ultimately the *grand mal* becomes well developed, and the attacks may then alternate.

(3) **Jacksonian Epilepsy.**—This is also known as cortical, symptomatic, or partial epilepsy. It is distinguished from the ordinary epilepsy by the important fact that consciousness is retained or is lost late. The attacks are usually the result of irritative lesions in the motor zone, though there are probably also sensory equivalents of this motor form. In a typical attack the spasm begins in a limited muscle group of the face, arm, or leg. The zygomatic muscles, for instance, or the thumb may twitch, or the toes may first be moved. Prior to the twitching the patient may feel a sensation of numbness or tingling in the part affected. The spasm extends and may involve the muscles of one limb only or of the face. The patient is conscious throughout and watches, often with interest, the march of the spasm.

The onset may be slow, and there may be time, as in a case which I have reported, for the patient to place a pillow on the floor, so as to be as comfortable as possible during the attack. The spasms may be localized for years, but there is a great risk that the partial epilepsy may become general. The condition is due, as a rule, to an irritative lesion in the motor zone. Thus of 107 cases analyzed by Roland, there were 48 of tumor, 21 instances of inflammatory softening, 14 instances of acute and chronic meningitis, and 8 cases of trauma. The remaining instances were due to hæmorrhage or abscess, or were associated with sclerosis cerebri. Two other conditions may be mentioned, which may cause typical Jacksonian epilepsy—namely, uræmia and progressive paralysis of the insane. A considerable number of the cases of Jacksonian epilepsy are found in children following hemiplegia, the so-called post-hemiplegic epilepsy. The convulsions usually begin on the affected side, either in the arm or leg, and the fit may be unilateral and without loss of consciousness. Ultimately they become more severe and general.

Diagnosis.—In major epilepsy the suddenness of the attack, the abrupt loss of consciousness, the order of the tonic and clonic spasm, and the relaxation of the sphincters at the height of the attack are distinctive features. The convulsive seizures due to uræmia are epileptic in character and usually readily recognized by the existence of greatly increased tension and the condition of the urine. Practically in young adults hysteria causes the greatest difficulty, and may closely simulate true epilepsy. The

following table from Gowers' work draws clearly the chief differences between them:

	Epileptic.	Hysteroid.
Apparent cause	none.	emotion.
Warning	any, but especially unilateral or epigastric auræ.	palpitation, malaise, choking, bilateral foot aura.
Onset	always sudden.	often gradual.
Scream	at onset.	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone.	rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	tongue.	lips, hands, or other people and things.
Micturition	frequent.	never.
Defecation	occasional.	never.
Talking	never.	frequent.
Duration	a few minutes.	more than ten minutes, often much longer.
Restraint necessary	to prevent accident.	to control violence.
Termination	spontaneous.	spontaneous or induced (water, etc.).

Recurring epileptic seizures in a person over thirty who has not had previous attacks is always suggestive of organic disease. According to H. C. Wood, whose opinion is supported by that of Fournier, in 9 cases out of 10 the condition is due to syphilis.

Petit mal must be distinguished from attacks of syncope, and the vertigo of Ménière's disease, of a cardiac lesion, and of indigestion. In these cases there is no actual loss of consciousness, which forms a characteristic though not an invariable feature of *petit mal.*

Jacksonian epilepsy has features so distinctive and peculiar that it is at once recognized. It is by no means easy, however, always to determine upon what the spasm depends. Irritation in the motor centres may be due to a great variety of causes, among which tumors and localized meningo-encephalitis are the most frequent; but it must not be forgotten that in uræmia localized epilepsy may occur. The most typical Jacksonian spasms also are not infrequent in general paresis of the insane.

Prognosis.—This may be given to-day in the words of Hippocrates: "The prognosis in epilepsy is unfavorable when the disease is congenital, and when it endures to manhood, and when it occurs in a grown person without any previous cause. . . . The cure may be attempted in young persons, but not in old."

Death during the fit rarely occurs, but it may happen if the patient falls into the water or if the fit comes on while he is eating. Occasionally the fits seem to stop spontaneously. This is particularly the case in the epilepsy in children which has followed the convulsions of teething or of the fevers. Frequency of the attacks and marked mental disturbance are unfavorable indications. Hereditary predisposition is apparently of no moment in the prognosis. The outlook is better in males than in females. The post-hemiplegic epilepsy is rarely arrested. Of the cases coming on

in adults, those due to syphilis and to local affections of the brain allow a more favorable prognosis.

Treatment.—*General.*—In the case of children the parents should be made to understand from the outset that epilepsy in the great majority of cases is an incurable affection, so that the disease may interfere as little as possible with the education of the child. The subjects need firm but kind treatment. Indulgence and yielding to caprices and whims are followed by weakening of the moral control, which is so necessary in these cases. The disease does not incapacitate a person for all occupation. It is much better for epileptics to have some definite pursuit. There are many instances in which they have been persons of extraordinary mental and bodily vigor, as, for example, Julius Cæsar and Napoleon. One of the most distressing features in epilepsy is the gradual mental impairment which follows in a certain number of cases. If such patients become extremely irritable or show signs of violence they should be placed under supervision in an asylum. Marriage should be forbidden to epileptics. During the attack a cork or bit of rubber should be placed between the teeth and the clothes should be loosened. The patient should be in the recumbent posture. As the attack usually passes off with rapidity, no special treatment is necessary, but in cases in which the convulsion is prolonged a few whiffs of chloroform or nitrite of amyl or a hypodermic of a quarter of a grain of morphia may be given.

Dietetic.—The old authors laid great stress upon regimen in epilepsy. The important point is to give the patient a light diet at fixed hours, and on no account to permit overloading of the stomach. Meat should not be given more than once a day. There are cases in which animal food seems injurious. A strict vegetable diet has been warmly recommended. The patient should not go to sleep until the completion of gastric digestion.

Medicinal.—The bromides are the only remedies which have a special influence upon the disease. Either the sodium or potassium salt may be given. Sodium bromide is probably less irritating and is better borne for a long period. It may be given in milk, in which it is scarcely tasted. In all instances the dilution should be considerable. In adults it is well taken in soda water or in some mineral water. The dose for an adult should be from half a drachm to a drachm and a half daily. As Seguin recommends, it is often best to give but a single dose daily, about four to six hours before the attacks are most likely to occur. For instance, in the case of nocturnal epilepsy a drachm should be given an hour or two after the evening meal. If the attack occurs early in the morning, the patient should take a full dose when he awakes. When given three times a day it is best given after meals. Each case should be carefully studied to determine how much bromide should be used. The individual susceptibility varies and some patients require more than others. Fortunately, children take the drug well and stand proportionately larger doses than adults. Saturation is indicated by certain unpleasant effects, particularly drowsiness, mental torpor, and gastric and cardiac distress. Loss of palate reflex is one of the earliest indications that the system is under the influence of the bromides, and is a condition which should be attained. A very unpleasant feature

is the development of acne, which, however, is no indication of bromism. Seguin states that the tendency to this is much diminished by giving the drug largely diluted in alkaline waters and administering from time to time full doses of arsenic. To be effectual the treatment should be continued for a prolonged period and the cases should be incessantly watched in order to prevent bromism. The medicine should be continued for at least two years after the cessation of the fits; indeed, Seguin recommends that the reduction of the bromides should not be begun until the patient has been three years without any manifestations. Written directions should be given to the mother or to the friends of the patient, and he should not himself be held responsible for the administration of the medicine. A book should be provided in which the daily number of attacks and the amount of medicine taken should be noted. The addition of belladonna to the bromide is warmly recommended by Black, of Glasgow. In very obstinate cases Flechsig uses opium, 5 or 6 grains, in three doses daily; then at the end of six weeks opium is stopped and the bromides in large amounts, 75 to 100 grains daily, are used for two months.

Among other remedies which have been recommended as controlling epilepsy are chloral, cannabis indica, zinc, nitroglycerin, and borax. Nitroglycerin is sometimes advantageous in *petit mal*, but is not of much service in the major form. To be beneficial it must be given in full doses, from 2 to 5 minims of the 1-per-cent solution, and increased until the physiological effects are produced. Counter-irritation is rarely advisable. When the aura is very definite and constant in its onset, as from the hand or from the toe, a blister about the part or a ligature tightly applied may stop the oncoming fit. In children, care should be taken that there is no source of peripheral irritation. In boys, adherent prepuce may occasionally be the cause. The irritation of teething, the presence of worms, and foreign bodies in the ears or nose have been associated with epileptic seizures.

The subjects of a chronic and, in most cases, a hopelessly incurable disease, epileptic patients form no small portion of the unfortunate victims of charlatans and quacks, who prescribe to-day, as in the time of the father of medicine, "purifications and spells and other illiberal practices of like kind."

Surgical.—In Jacksonian epilepsy the propriety of surgical interference is universally granted. It is questionable, however, whether in the epilepsy following hemiplegia, considering the anatomical condition, it is likely to be of any benefit. In idiopathic epilepsy, when the fit starts in a certain region—the thumb, for instance—and the signal symptom is invariable, the centre controlling this part may be removed. This procedure has been practised by Macewen, Horsley, Keen, and others, but time alone can determine its value. The traumatic epilepsy, in which the fit follows fracture, is much more hopeful.

The operation, *per se*, appears in some cases to have a curative effect. Thus of 50 cases of trephining for epilepsy in which nothing abnormal was found to account for the symptoms, 25 were reported as cured and 18 as improved. The operations have not been always on the skull, and White has collected an interesting series in which various surgical procedures have

been resorted to, often with curative effect, such as ligation of the carotid artery, castration, tracheotomy, excision of the superior cervical ganglia, incision of the scalp, circumcision, etc.

VII. MIGRAINE (*Hemicrania; Sick Headache*).

Definition.—A paroxysmal affection characterized by severe headache, usually unilateral, and often associated with disorders of vision.

Etiology.—The disease is frequently hereditary and has occurred through several generations. Women and the members of neurotic families are most frequently attacked. It is an affection from which many distinguished men have suffered and have left on record an account of the disease, notably the astronomer Airy. Edward Liveing's work is the standard authority upon which most of the subsequent articles have been based. A gouty or rheumatic taint is present in many instances. Sinkler has called special attention to the frequency of reflex causes. Migraine has long been known to be associated with uterine and menstrual disorders. Nutritive disturbances are common, and attempts have been made by Haig and others to associate the attacks with disturbed uric-acid output. Certainly the amount of uric acid excreted just prior to and during an attack is reduced. Others regard the disease as a toxæmia from disordered intestinal digestion. Many of the headaches from eye-strain are of the hemicranial type. Brunton refers to caries of the teeth as a cause of these headaches, even when not associated with toothache. Cases have been described in connection with adenoid growths in the pharynx, and particularly with abnormal conditions of the nose. Many of the attacks of severe headaches in children are of this nature, and the eyes and nostrils should be examined with great care. Sinkler refers to a case in a child of two years, and Gowers states that a third of all the cases begin between the fifth and tenth years of age. The direct influences inducing the attack are very varied. Powerful emotions of all sorts are the most potent. Mental or bodily fatigue, digestive disturbances, or the eating of some particular article of food may be followed by the headache. The paroxysmal character is one of the most striking features, and the attacks may recur on the same day every week, every fortnight, or every month. Headaches of the migraine type may recur for years in connection with chronic Bright's disease.

Symptoms.—Premonitory signs are present in many cases, and the patient can tell when an attack is coming on. Remarkable prodromata have been described, particularly in connection with vision. Apparitions may appear—visions of animals, such as mice, dogs, etc. Transient hemianopia or scotoma may be present. In other instances there is spasmodic action of the pupil on the affected side, which dilates and contracts alternately, the condition known as *hippus*. Frequently the disturbance of vision is only a blurring, or there are balls of light, or zigzag lines, or the so-called fortification spectra (teichopsia), which may be illuminated with gorgeous colors. Disturbances of the other senses are rare. Numbness of the tongue and face and occasionally of the hand may occur with tingling.

More rarely there are cramps or spasms in the muscles of the affected side. Transient aphasia has also been noted. Some patients show marked psychical disturbance, either excitement or, more commonly, mental confusion or great depression. Dizziness occurs in some cases. The headache follows a short time after the prodromal symptoms have appeared. It is cumulative and expansile in character, beginning as a localized small spot, which is generally constant either on the temple or forehead or in the eyeball. It is usually described as of a penetrating, sharp, boring character. At first unilateral, it gradually spreads and involves the side of the head, sometimes the neck, and the pains may pass into the arm. In other cases both sides are affected. Nausea and vomiting are common symptoms. If the attack comes on when the stomach is full, vomiting usually gives relief. Vasomotor symptoms may be present. The face, for instance, may be pale, and there may be a marked difference between the two sides. Subsequently the face and ear on the affected side may become a burning red from the vasodilator influences. The pulse may be slow. The temporal artery on the affected side may be firm and hard, and in a condition of arterio-sclerosis—a fact which has been confirmed anatomically by Thoma. Few affections are more prostrating than migraine, and during the paroxysm the patient may scarcely be able to raise the head from the pillow. The slightest noise or light aggravates the condition.

The duration of the entire attack is variable. The severer forms usually incapacitate the person for at least three days. In other instances the entire attack is over in a day. The disease recurs for years, and in cases with a marked hereditary tendency may persist throughout life. In women the attacks often cease after the climateric, and in men after the age of fifty. Two of the greatest sufferers I have known, who had recurring attacks every few weeks from early boyhood, now have complete freedom.

The nature of the disease is unknown. Liveing's view, that it is a nerve storm or form of periodic discharge from certain sensory centres and is related to epilepsy, has found much favor. According to this view, it is the sensory equivalent of a true epileptic attack. Mollendorf, Latham, and others regard it as a vaso-motor neurosis, and hold that the early symptoms are due to vaso-constrictor and the later symptoms to vaso-dilator influences. The fact of the development of arterio-sclerosis in the arteries of the affected side is a point of interest bearing upon this view.

Treatment.—The patient is fully aware of the causes which precipitate an attack. Avoidance of excitement, regularity in the meals, and moderation in diet are important rules. I have known cases greatly benefitted by a strict vegetable diet. The treatment should be directed toward the removal of the conditions upon which the attacks depend. In children much may be done by watchfulness and care on the part of the mother in regulating the bowels and watching the diet of the child. Errors of refraction should be adjusted. On no account should such children be allowed to compete in school for prizes. A prolonged course of bromides sometimes proves successful. If anæmia is present, iron and arsenic should be given. When the arterial tension is increased a course of nitroglycerin may be tried. Not too much, however, should be expected of the preventive treat-

ment of migraine. It must be confessed that in a very large proportion of the cases the headaches recur in spite of all we can do. Herter advises, so soon as the patient has any intimation of the attack, to wash out the stomach with water at 105°, and to give a brisk saline cathartic. During the paroxysm the patient should be kept in bed and absolutely quiet. If the patient feels faint and nauseated, a small cup of hot, strong coffee or 20 drops of chloroform give relief. Cannabis indica is probably the most satisfactory remedy. Seguin recommends a prolonged course of the drug. Antipyrin, antifebrin, and phenacetin have been much used of late. When given early, at the very outset of the paroxysm, they are sometimes effective. The doses which have been recommended of antifebrin and antipyrin are often dangerous, and I have seen in a case of migraine unpleasant collapse symptoms follow a 25-grain dose of antipyrin which the patient had taken on her own responsibility. Smaller, repeated doses are more satisfactory. Of other remedies, caffeine, in 5-grain doses of the citrate, nux vomica, and ergot have been recommended. Electricity does not appear to be of much service.

VIII. NEURALGIA.

Definition.—A painful affection of the nerves, due either to functional disturbance of their central or peripheral extremities or to neuritis in their course.

Etiology.—Members of neuropathic families are most subject to the disease. It affects women more than men. Children are rarely attacked. Of all causes, debility is the most frequent. It is often the first indication of an enfeebled nervous system. The various forms of anæmia are frequently associated with neuralgia. It may be a prominent feature at the onset of certain acute diseases, particularly typhoid fever. Malaria is believed to be a potent cause, but it has not been shown that neuralgia is more frequent in malarial districts, and the error has probably arisen from regarding periodicity as a special manifestation of paludism. It occasionally occurs in malarial cachexia. Exposure to cold is a cause in very susceptible persons. Reflex irritation, particularly from carious teeth, may induce neuralgia of the fifth nerve. The disease occurs sometimes in rheumatism, gout, lead poisoning, and diabetes. Persistent neuralgia may be a feature of latent Bright's disease.

Symptoms.—Before the onset of the pain there may be uneasy sensations, sometimes tingling in the part which will be affected. The pain is localized to a certain group or division of nerves, usually affecting one side. The pain is not constant, but paroxysmal, and is described as stabbing, burning, or darting in character. The skin may be exquisitely tender in the affected region, particularly over certain points along the course of the nerve, the so-called tender points. Movements, as a rule, are painful. Trophic and vaso-motor changes may accompany the paroxysm; the skin may be cool, and subsequently hot and burning; occasionally local œdema or erythema occurs. More remarkable still are the changes in the hair, which may become blanched (canities), or even fall out. Fortunately,

And lastly, in obstinate cases an ordinary tape seton may be inserted through the skin at the back of the neck, to be worn for three months, a plan of treatment which has the strongest possible recommendation from Mr. Whitehead, of Manchester.

such alterations are rare. Twitchings of the muscles, or even spasms, may be present during the paroxysm. After lasting a variable time—from a few minutes to many hours—the attack subsides. Recurrence may be at definite intervals—every day at the same hour, or at intervals of two, three, or even seven days. Occasionally the paroxysms develop only at the catamenia. This periodicity is quite as marked in non-malarial as in malarial regions.

Clinical Varieties, depending on the Nerve Groups affected.—(1) *Trifacial Neuralgia; Tic Douloureux; Prosopalgia.*—All the branches are rarely involved together. The ophthalmic is most often affected, but in severe attacks the pains, though more intense in one division, radiate over the other branches. At the outset there may be hyperæsthesia of the skin and sensitiveness of the mucous membrane. Pressure is painful at the points of emergence of the nerve trunk, and where the nerves enter the muscles. Sometimes in addition, as Trousseau pointed out, there are pains at the occipital protuberance and in the upper cervical spines. When the ophthalmic division is affected the eye may weep and the conjunctivæ are injected and painful. In the upper maxillary division there is a tender point where the nerve leaves the infraorbital canal, and the pain is specially marked along the upper teeth. In the lower branches, which are more frequently involved, there are painful points along the auriculo-temporal nerve and the pain radiates in the region of the ear along the lower jaw and teeth. The movements of mastication and speaking may be painful. Salivation is not uncommon. Herpes may occur about the eye or the lips. In protracted cases there may be atrophy or induration of the skin. Some of the forms of facial neuralgia are of frightful intensity and the recurring attacks render the patient's life almost insupportable.

(2) *Cervico-occipital neuralgia* involves the posterior branches of the first four cervical nerves, particularly the inferior occipital, at the emergence of which there is a painful point about half-way between the mastoid process and the first cervical vertebra. It may be caused by cold, and these nerves are often affected in cervical caries.

(3) *Cervico-brachial neuralgia* involves the sensory nerves of the brachial plexus, particularly in the cubital division. When the circumflex nerve is involved the pain is in the deltoid. The pain is most commonly about the shoulder and down the course of the ulnar nerve. There is usually a marked tender point upon this nerve at the elbow. This form rarely follows cold, but more frequently results from rheumatic affections of the joints, and trauma.

(4) *Neuralgia of the phrenic nerve* is rare. It is sometimes found in pleurisy and in pericarditis. The pain is chiefly at the lower part of the thorax on a line with the insertion of the diaphragm, and here may be painful points on deep pressure. Full inspiration is painful, and there is great sensitiveness on coughing or in the performance of any movement by which the diaphragm is suddenly depressed.

(5) *Intercostal Neuralgia.*—Next to the *tic douloureux* this is the most important form. It is most frequent in women and very common in hysteria and anæmia. The pain in caries and aneurism is felt in the intercostal

nerves. They are also the seat of the intense pain in inflammation of the pleura. The pain is often constant and exaggerated by movements. Pleurodynia is supposed by some to be local intercostal neuralgia, confined to one spot, usually along the course or at the exit of the nerves. Herpes zoster or zona occurs with the most aggravated form of intercostal neuralgia. The pain usually precedes the eruption, which consists of a series of pearly vesicles, which take two or three days to develop and gradually disappear. The eruption may occur without much pain. The most distressing feature in the complaint is the persistence in the pain after the eruption has subsided. The eruption and the neuralgia are in reality manifestations of neuritis. Changes have been found in the nerves and in the ganglia of the dorsal roots. The pain of zona may persist indefinitely, and it has been known to be so intractable that in despair the person has committed suicide.

(6) *Lumbar Neuralgia.*—The affected nerves are the posterior fibres of the lumbar plexus, particularly the ilio-scrotal branch. The pain is in the region of the iliac crest, along the inguinal canal, in the spermatic cord, and in the scrotum or labium majus. The affection known as irritable testis, probably a neuralgia of this nerve, may be very severe and accompanied by syncopal sensations.

(7) *Coccydynia.*—This is regarded as a neuralgia of the coccygeal plexus. It is most common in women, and is aggravated by the sitting posture. It is very intractable, and may necessitate the removal of the coccyx, an operation, however, which is not always successful. Neuralgias of the nerves of the leg have already been considered.

(8) *Neuralgias of the Nerves of the Feet.*

Painful Heel.—Both in women and men there may be about the heel severe pains which interfere seriously with walking—the pododynia of S. D. Gross. There may be little or no swelling, no discoloration, and no affection of the joints.

Plantar Neuralgia.—This is often associated with a definite neuritis, such as follows typhoid fever, and has been seen in an aggravated form in caisson disease (Hughes). The pain may be limited to the tips of the toes or to the ball of the great toe. Numbness, tingling, and hyperæsthesia or sweating may occur with it. Following the cold-bath treatment in typhoid fever it is not uncommon for patients to complain of great sensitiveness in the toes.

Metatarsalgia.—Morton's (Thomas G.) "painful affection of the fourth metatarso-phalangeal articulation" is a peculiar and very trying disorder, seen most frequently in women, and usually in one foot. Morton regards it as due to a pinching of the metatarsal nerve. The disease rarely gets well without operation. The red, painful neuralgia—erythromelalgia—is described under the vaso-motor and trophic disturbances.

(9) *Visceral Neuralgias.*—The more important of these have already been referred to in connection with the cardiac and the gastric neuroses. They are most frequent in women, and are constant accompaniments of neurasthenia and hysteria. The pains are most common in the pelvic region, particularly about the ovaries. Nephralgia is of great interest, for,

as has already been mentioned, the symptoms may closely simulate those of stone.

Treatment.—Causes of reflex irritation should be carefully removed. The neuralgia, as a rule, recurs unless the general health improves; so that tonic and hygienic measures of all sorts should be employed. Often a change of air or surroundings will relieve a severe neuralgia. I have known obstinate cases to be cured by a prolonged residence in the mountains, with an out-of-door life and plenty of exercise. A strict vegetable diet will sometimes relieve the neuralgia or headache of a gouty person. Of general remedies, iron is often a specific in the cases associated with chlorosis and anæmia. Arsenic, too, is very beneficial in these forms, and should be given in ascending doses. The value of quinine has been much overrated. It probably has no more influence than any other bitter tonic, except in the rare instances in which the neuralgia is definitely associated with malarial poisoning. Strychnine, cod-liver oil, and phosphorus are also advantageous. Of remedies for the pain, the new analgesics should first be tried—antipyrin, antifebrin, and phenacetin—for they are sometimes of service. Morphia should be given with great caution, and only after other remedies have been tried in vain. On no consideration should the patient be allowed to use the hypodermic syringe. Gelsemium is highly recommended. Of nervine stimulants, valerian and ether, which often act well together, may be given. Alcohol is a valuable though dangerous remedy, and should not be ordered for women. In the trifacial neuralgia nitroglycerin in large doses may be tried. Aconitia in doses of from one two-hundredth to one one-hundred-and-fiftieth of a grain may be tried. In gouty and rheumatic subjects cannabis indica and cimicifuga are recommended with the lithium salts.

Of local applications, the thermo-cautery is invaluable, particularly in zona and the more chronic forms of neuralgia. Acupuncture may be used, or aquapuncture, the injection of distilled water beneath the skin. Chloroform liniment, camphor and chloral, menthol, the oleates of morphia, atropia, and belladonna used with lanolin may be tried. Freezing over the tender point with ether spray is sometimes successful. The continuous current may be used. The sponges should be warm, and the positive pole should be placed near the seat of the pain. The strength of the current should be such as to cause a slight tingling or burning, but not pain.

The surgical treatment of intractable neuralgia embraces nerve stretching and excision. The latter is the more satisfactory, but too often the pain returns.

IX. PROFESSIONAL SPASMS; OCCUPATION NEUROSES.

The continuous and excessive use of the muscles in performing a certain movement may be followed by an irregular, involuntary spasm or cramp, which may completely check the performance of the action. The condition is found most frequently in writers, hence the term writer's cramp or scrivener's palsy; but it is also common in piano and violin players and

in telegraph operators. The spasms occur in many other persons, such as milkmaids, weavers, and cigarette-rollers.

The most common form is writer's cramp, which is much more frequent in men than in women. Of 75 cases of impaired writing power reported by Poore, all of the instances of undoubted writer's cramp were in men. Morris J. Lewis states that in this country, in the telegrapher's cramp, women, who are employed a great deal in telegraphy, are much less frequently affected (only 4 out of 43 cases). Persons of a nervous temperament are more liable to the disease. Occasionally it follows slight injury.

Gowers states that in a majority of the cases a faulty method of writing has been employed, using either the little finger or the wrist as the fixed point. Persons who write from the middle of the forearm or from the elbow are rarely affected.

No anatomical changes have been found. The most reasonable explanation of the disease is that it results from a deranged action of the nerve centres presiding over the muscular movements involved in the act of writing, a condition which has been termed irritable weakness. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this lessened resistance be carried too far, there is an increase and irregular discharge of nerve energy, which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves" (Gay).

Symptoms.—These may be described under five heads (Lewis).

(*a*) *Cramp* or *Spasm.*—This is often an early symptom and most commonly affects the forefinger and thumb; or there may be a combined movement of flexion and adduction of the thumb, so that the pen may be twisted from the grasp and thrown to some distance. Weir Mitchell has described a lock-spasm, in which the fingers become so firmly contracted upon the pen that it cannot be removed.

(*b*) *Paresis* and *Paralysis.*—This may occur with the spasm or alone. The patient feels a sense of weakness and debility in the muscles of the hand and arm and holds the pen feebly. Yet in these circumstances the grasp of the hand may be strong and there may be no paralysis for ordinary acts.

(*c*) *Tremor.*—This is most commonly seen in the forefinger and may be a premonitory symptom of atrophy. It is not an important symptom, and is rarely sufficient to produce disability.

(*d*) *Pain.*—Abnormal sensations, particularly a tired feeling in the muscles, are very constantly present. Actual pain is rare, but there may be irregular shooting pains in the arm. Numbness or soreness may exist. If, as sometimes happens, a subacute neuritis develops, there may be pain over the nerves and numbness or tingling in the fingers.

(*e*) *Vaso-motor Disturbances.*—These may occur in severe cases. There may be hyperæsthesia. Occasionally the skin becomes glossy, or there is a condition of local asphyxia resembling chilblains. In attempting to write, the hand and arm may become flushed and hot and the veins increased in size. Early in the disease the electrical reactions are normal, but in advanced cases there may be diminution of faradic and sometimes increase in the galvanic irritability.

Diagnosis.—A well-marked case of writer's cramp or palsy could scarcely be mistaken for any other affection. Care must be taken to exclude the existence of any cerebro-spinal disease, such as progressive muscular atrophy or hemiplegia. The physician is sometimes consulted by nervous persons who fancy they are becoming subject to the disease and complain of stiffness or weakness without displaying any characteristic features.

Prognosis.—The course of the disease is usually chronic. If taken in time and if the hand is allowed perfect rest, the condition may improve rapidly, but too often there is a strong tendency to recurrence. The patient may learn to write with the left hand, but this also may after a time be attacked.

Treatment.—Various prophylactic measures have been advised. As mentioned, it is important that a proper method of writing be adopted. Gowers suggests that if all persons wrote from the shoulder writer's cramp would practically not occur. Various devices have been invented for relieving the fatigue, but none of them are very satisfactory. The use of the type-writer has diminished very much the frequency of scrivener's palsy. Rest is essential. No measures are of value without this. Massage and manipulation, when combined with systematic gymnastics, give the best results. Poore recommends the galvanic current applied to the muscles, which are at the same time rhythmically exercised. In very obstinate cases the condition remains incurable. I saw a few years ago a distinguished gynæcologist who had had writer's cramp twenty years before, and who had all sorts of treatment, including the Wolff's method, without any avail. He still has it in aggravated form, but he can do all the finer manipulations of operative work without any difficulty.

The nutrition of the patients is apt to be much impaired, and cod-liver oil, strychnia, and other tonics will be found advantageous. Local applications are of little benefit. Tenotomy and nerve-stretching have been abandoned.

X. TETANY.

Definition.—An affection characterized by peculiar bilateral tonic spasms, either paroxysmal or continued, of the extremities.

Etiology.—The disease occurs under very different conditions, of which the following may be recognized:

(*a*) Epidemic tetany, also known as rheumatic tetany. In certain parts of the continent of Europe the disease has prevailed widely, particularly in the winter season. Von Jaksch, who has described an epidemic

form occurring in young men of the working classes, sometimes with slight fever, regards the disease as infectious. This form is acute, lasting only two or three weeks and rarely proving fatal.

(*b*) A majority of the cases are found in association with debility following lactation and chronic diarrhœa, or in the malnutrition of rickets. From its occurrence in nursing women Trousseau called it nurse's contracture. It may also develop during pregnancy or recur in successive pregnancies. It has been found as a sequence of the acute fevers, and in some typhoid epidemics many cases have occurred.

(*c*) Tetany may follow removal of the thyroid gland. Thirteen cases, for example, followed 78 operations on enlarged thyroid in Billroth's clinic, and 6 of them proved fatal. James Stewart has reported an instance in which with the tetany there were symptoms of myxœdema, and no trace of the thyroid gland. Removal of the thyroid in dogs is followed by tetany.

(*d*) And, lastly, there is a form of tetany which is associated with dilatation of the stomach, particularly after the organ has been washed out.

On this continent true tetany is an extremely rare disease. Griffith has collected 72 cases, among which, however, cases of carpo-pedal spasm are included.

The nature of the disease is unknown; certain forms depend undoubtedly on loss of the function of the thyroid gland.

Symptoms.—In cases associated with general debility or in children with rickets the spasm is limited to the hands and feet. The fingers are bent at the metacarpo-phalangeal joint, extended at the terminal joints, pressed close together, and the thumb is contracted in the palm of the hand. The wrist is flexed, the elbows are bent, and the arms are folded over the chest. In the lower limbs the feet are extended and the toes adducted. The muscles of the face and neck are less commonly involved, but in severe cases there may be trismus, and the angles of the mouth are drawn out. The skin of the hands and feet is sometimes tense and œdematous. The spasms are usually paroxysmal and last for a variable time. In children the attack may pass off in a few hours. In some of the severer chronic cases in adults the stiffness and contracture may continue or even increase for many days, and the attack may last as long as two weeks. In the acute cases the temperature may be elevated and the pulse quickened. In the severe paroxysms there may be involvement of the muscles of the back and of the thorax, inducing dyspnœa and cyanosis. Certain additional features, valuable in diagnosis, are present.

Trousseau's symptom: "So long as the attack is not over, the paroxysms may be reproduced at will. This is effected by simply compressing the affected parts, either in the direction of their principal nerve trunks or over their blood-vessels, so as to impede the venous or arterial circulation."

Chovstek's symptom is shown in the remarkable increase in the mechanical excitability of the motor nerves. A slight tap, for example, in the course of the facial nerve will throw the muscles to which it is distributed into active contraction. Erb has shown that the electrical irritability of the nerves is also greatly increased, and Hofmann has demon-

Tetany in Infancy; with a Report of Six Cases

Presented to the Section on Diseases of Children, at the Forty-ninth Annual Meeting of the American Medical Association, held at Denver, Colo., June 7-10, 1898.

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REPRINTED FROM
THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.
NOVEMBER 5, 1898.

CHICAGO:
AMERICAN MEDICAL ASSOCIATION PRESS.
1898.

TETANY IN INFANCY; WITH A REPORT OF SIX CASES.

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Very great differences of opinion exist with regard to what constitutes tetany, as well as to the frequency of its occurrence and its etiology and pathology.

It is generally accepted that the most characteristic symptom of tetany is muscular rigidity, this rigidity occurring as intermittent paroxysmal contractures. The duration and intensity of the contractures vary, in some cases being long and remittent, in others fleeting and occurring at long intervals. The extent of the contractures also varies within wide limits. the seat par excellence, however, being in the muscles of the arms, especially of the fore-arms. The positions assumed by the hands and feet during these contractures are very characteristic. The fingers are flexed at the metacarpo-phalangeal joints, while the phalanges are extended. The thumb is strongly adducted in the extended position. The wrist is acutely flexed and the hand turned to the ulnar side. The elbow- and shoulder-joints are not involved in the milder cases. The feet are strongly extended, sometimes in the position of pure equinus, but more often in that of equino-varus. The first phalanges of the toes are strongly flexed, the others extended. The knee- and hip-joints are usually free. Opisthotonos is not very uncommon in the severe cases. Associated with the contractures are other symptoms, some or all of which are present in every case. These are: Increased electric excitability of both nerve and muscle to faradism and galvanism, with changes in the qualitative reaction to galvanism. This is often known as Erb's symptom and is probably the most constant. Increased

2

mechanical excitability of both nerve and muscle. Special examples of this are the occurrence of characteristic contractures as the result of pressure on the large nerve trunks and arteries, "Trousseau's symptom," and spasm of the facial muscles when the skin over the trunk of the facial nerve is irritated or the nerve is struck a sharp blow, "Chvostek's symptom" or the "facialis phenomenon." Laryngospasm. Convulsions. Sensory disturbances, including pain on motion and during spasms, as well as many others impossible to determine in infants. Vasomotor and trophic disturbances, the most characteristic being edema of the wrists and ankles. Fever, which is inconstant and intermittent. The intelligence is always unimpaired, even during the paroxysmal contractures.

All agree that the presence of the peculiar paroxysmal contractures justifies the diagnosis of tetany. There is great difference of opinion, however, as to whether the presence of one or more of the other symptoms without contractures justifies the diagnosis. It is to this condition that the term "latent tetany" is applied. Even here, however there is no consensus of opinion, some apparently limiting the term to the condition in which irritation of the muscle or nerve brings on contractures which are otherwise absent, while others consider the presence of even one symptom as pathognomonic and sufficient for the diagnosis. As a rule, those who recognize the existence of latent tetany consider it the same as tetany, differing from the latter only in degree. If these symptoms are to be considered as pathognomonic and sufficient for the diagnosis of tetany, whether the characteristic contractures are present or not, they should be always present where there are contractures and should never occur in other conditions. Trousseau's symptom and the facial phenomenon are sometimes absent, and often only temporarily present when there are typic contractures. Moreover, Trousseau's symptom is often met with in hysteria, while the facial phenomenon is found in many other conditions, as epilepsy, hysteria,

phthisis and neurasthenia. Laryngospasm is frequent in tetany, but not constant, and is often seen in children who have no other symptoms of tetany. Convulsions and disturbances of sensation, as well as vasomotor and trophic disturbances, occur in many other diseases. It is evident, therefore, that these three symptoms—laryngospasm, Trousseau's symptom and the facial phenomenon—are not constant in cases which show the typic contractures of tetany, and that they occur in many conditions in which there is no question of tetany. Hence they can not be considered as pathognomonic of tetany, and do not, either singly or in combination, afford sufficient ground for the diagnosis either of tetany or latent tetany. They must be regarded merely as particular instances of increased mechanical or reflex excitability which may be the result of a very great variety of causes and pathologic conditions. The only pathognomonic symptom of tetany, therefore, is spontaneous, intermittent, paroxysmal muscular contracture. The term "tetany" should be applied only to those cases in which this symptom is present, and no cases of increased reflex excitability in which this symptom does not occur should be regarded as examples of the disease. The term "latent tetany," is therefore a misnomer and should be dropped.

ETIOLOGY.

Numerous theories as to the etiology of tetany have been advanced, and have found vigorous defenders and opponents. The question is still unsettled, however, probably partly on account of the differences of opinion as to what constitutes tetany, and partly on account of the inherent difficulties of the subject. The theory of the older authors that it is of rheumatic origin has long been given up. The position that it is merely a manifestation of hysteria must also be regarded as untenable. The theory that it is cerebral in origin was founded on an erroneous conception of the disease, and is no longer accepted. The theory that it is a variety of acute anterior poliomyelitis can

4

not easily be set aside, however, as certain quite definite lesions have been found in the anterior horns. As the cases of tetany which do not die of some intercurrent disease nearly always recover, these lesions must, as a rule, be transitory. They may very probably be frequently due to the action of toxic substances in the circulation, the result of diseased conditions elsewhere.

The most ardent and extreme advocate of the rachitic theory is undoubtedly Kassowitz. His line of argument is somewhat as follows: Craniotabes is a lesion of rickets. Craniotabes may be represented only by meningeal hyperemia, and hence be incapable of demonstration. Laryngospasm is due to craniotabes. Laryngospasm is a symptom of rickets. Other symptoms of increased neuro-muscular excitability are seen in rickets. They are of the same nature as laryngospasm and hence due to the same cause. Laryngospasm and other nervous manifestations are symptoms of tetany; in fact, they are tetany. Hence tetany is a symptom of rickets.

There are many obvious objections to this reasoning. It is not proven that craniotabes is the cause of laryngospasm, or that meningeal hyperemia produces the same results as craniotabes. Moreover, laryngospasm occurs in tetany when there is no evidence of rickets, in rickets when there are no other evidences of tetany, and alone when there are no evidences of either tetany or rickets.

Although not agreeing with the extreme views of Kassowitz, most of the German and Austrian authorities have accepted the rachitic theory in a more or less modified form. Some of the arguments in favor of this view are, that almost all cases of tetany show evidences of rickets, that they are both more frequent in winter, and that they occur in children of the same age. Some of the arguments against it are, that very many cases of other diseases than tetany show evidences of rickets, that the symptoms of increased neuro-muscular excitability, with the exception of

laryngospasm, are rare among rachitics, and that tetany is more likely to occur in mild than in severe cases of rickets. The fact that rickets is common all the year round, although much more so in the winter, while tetany very rarely occurs in the summer, would seem to point to some common cause for both conditions which is active only in the winter. This cause is most probably the indoor life of the winter, with its lack of sunlight and oxygen and its exposure to noxious vapors. These furnish the opportunity for the formation of respiratory toxins which may produce changes in the circulation that lead to malnutrition of various tissues with subsequent symptoms.

Several theories locating the origin of tetany in the gastro-intestinal canal have been advanced. These have received their main support from the French authorities. The dehydration and reflex theories need be mentioned only to be set aside. That of auto-intoxication, however, is worthy of careful consideration. The friends of this theory say that tetany is almost always associated with diarrhea or other digestive disturbances. They say that the reason that all children suffering from indigestion do not have tetany is that some peculiar chemic combination is necessary, or because their nervous system is insusceptible. When tetany occurs in infants who present no symptoms of indigestion, they assume that the nervous system is easily affected, or that this peculiar chemic combination may be produced without causing other symptoms. They also state that when the digestive disturbances are relieved the tetany ceases. They attribute the frequency of tetany in rachitis to the gastro-enteric disturbances of this disease. The opponents of this theory say that gastro-enteric disorders are very common and tetany very rare; that digestive disturbances are more common in summer and tetany in winter; and that the only basis for the assumption of peculiar susceptibility or special chemic combinations is in the imaginations of the originators.

6

The most recent, and perhaps the most plausible, theory assumes that there is no single pathologic cause for tetany, but that, like epilepsy, it may arise from many causes. Like epilepsy, too, it must be regarded merely as a nosologic entity, and not as a definite disease. In improper hygienic surroundings, in rickets, in gastro-intestinal disorders, in acute disease, and in various intoxications are present conditions capable of causing the formation of various toxic substances. The action of all these poisonous substances may show itself by a special modification, rather functional than organic, of the central or peripheral nervous system. The various lesions of the nervous system found in tetany are not inconsistent with this conception.

MORBID ANATOMY.

The lesions which have been found in the nervous system in tetany are very variable. Tonnellé, Blondeau, Grisolle, Trousseau and Bouchut have found hyperemia of various portions of the brain and cord and their meninges; Weiss, Bonome, Cervesato and Sarbo various degrees of poliomyelitis of the cervical enlargement; Stasse, Niemayer and Schultze peripheral neuritis; and Loos, Berger and others no lesions. The most characteristic lesions, however, are probably those in the cells of the anterior horns in the cervical enlargement. These vary in degree from hyperemia and swelling to atrophy and sclerosis, the severity of the process probably depending on the duration and intensity of the disease. It is probable that certain lesions in this region are present in all cases, but that in recent ones they are too superficial to be recognized. Whether they are primary here or secondary to an ascending neuritis is at present entirely undetermined. As they have not been found constantly, however, further verification is necessary before they can be accepted as the essential lesions of the disease. Nevertheless, they do correspond more closely to the symptoms of the disease than any other

lesions which have been described, and seem at any rate to afford a fairly satisfactory explanation for them.

FREQUENCY.

The frequency of tetany must naturally depend on the definition of the disease which is accepted. If the presence of symptoms of increased neuromuscular excitability is considered as evidence of latent tetany, and latent tetany is classed as tetany, the disease will be frequently encountered. If, however, only those cases in which the typic paroxysmal contractures are present are regarded as tetany it will be seen much less often. This difference in classification undoubtedly explains to a great extent the much greater frequency of the disease in Germany and Austria than in France and Italy. It does not entirely account for the discrepancy, however, and there is probably but little doubt that tetany, even in the strict interpretation of the term, is much more common in the first-named countries. It is still less common in this country, being, indeed, something of a rarity. Griffith, in addition to five cases of his own, was able to find but seventy-two cases reported in American literature up to 1894, although he included in his list a number of cases of muscular spasm, which, strictly speaking can not be accepted as cases of tetany. In a careful review of American medical literature from Jan. 1, 1894, to Jan. 1, 1898, I am able to find but thirteen additional cases. They are, in brief, as follows:

1. Lewi: Nine months. Constant, typic spasm in hands. Arms and legs painful. One convulsion. Spasm became remittent, then ceased. Duration, three weeks. Neurotic family history. Rachitic. No craniotabes. No gastro-intestinal disturbances.

2. Lewi: Eight months. Typic, intermittent spasms in hands and feet, lasting hours or days. Hands and feet tender and painful. Increased electric reaction. Duration, six weeks. Recurrent attack two months later with death in convulsion. An older child had had disease. Rachitic. No craniotabes. Gastro intestinal disturbance always. Constipation during attack.

3. Lewi: Eight months. Typic, continuous spasm in hands

and feet. Hands and feet swollen, tender and painful. Increased electric reaction. Duration, two weeks. Rachitic. No craniotabes. Vomiting. Constipation.

4. Lewi: Thirteen months. Remittent spasm in hands and feet, most in hands. Hands and feet swollen, tender and painful. Laryngismus stridulus. Duration, two weeks. An older child had had disease. Markedly rachitic. No craniotabes. Vomiting. Constipation.

5. Crandall: Six months. Began with peculiar attacks of staring. In a month well-marked and typic paroxysms, involving neck and all extremities, lasting one and a half to two hours. Five to eight attacks in twenty four hours. No laryngismus stridulus. Duration, eight weeks. Moderately rachitic. No craniotabes. Poor breast feeding. Diarrhea, later constipation. Phimosis. Correction of phimosis had no effect. Complete recovery in eleven days after correction of diet and relief of constipation.

6. Crandall: Eleven months. Twelve to twenty-four attacks of laryngismus stridulus and convulsions in twenty-four hours. Duration, two minutes. Spasms fairly typic. Duration, three weeks. Markedly rachitic. No craniotabes. Breast and general diet. Vomiting. Alternate diarrhea and constipation. Recovery in two days after correction of diet and cathartic.

7. Preston: Three and a half years. Typic spasms in arms, less so in legs, lasting a few minutes; intervals of months or ten to fifteen in twenty-four hours. No pain. Trousseau's symptom present. Began at one year. Duration unknown.

8. Krauss: Six months. Typic paroxysms; continuous for two weeks at height of attack. Much pain. Swelling of face and extremities. Trousseau's symptom present. Duration, five weeks. No note as to rickets. Artificial food. Constipation, then diarrhea. Dentition.

9. Krauss: Eighteen months. Mild attacks, lasting a few minutes, at intervals of half an hour to two hours. Duration, one week. No note as to rachitis. Vomiting. Diarrhea. Bronchitis.

10. Krauss: Thirteen months. Mild attacks, lasting five to ten minutes, several times, daily. Duration, sixteen days. No note as to rachitis. Gastro-intestinal disturbance. Bronchitis.

11. Krauss: Nine months. Paroxysms, involving hands and legs. Duration, three weeks. No note as to rachitis. Bronchitis.

12. Krauss: Eleven months Remittent paroxysms; most severe in arms. Duration, two weeks. No note as to rachitis. Severe gastro-intestinal disturbance. Dentition. Bronchitis.

13. Carpenter: Eleven months. Tonic spasms. Laryngismus stridulus. Recovery, slow. Mild recurrence for several years. No note as to rachitis. Followed purulent effusion in shoulder-joint after scarlet fever.

A case reported by Miles as tetany, and another by Carpenter, are probably cases of tetanus. In another, reported by Carpenter, the diagnosis was based entirely on laryngismus stridulus, and, therefore, can not be accepted.

The disease is certainly a very unusual one in Boston, as is shown by the following statistics from the Medical Out-Patient Department of the Infants' Hospital, during the last two years. The great majority of the patients treated there are infants under two years of age, belonging to the poorest classes. Both their age and hygienic surroundings would seem likely, therefore, to render them especially favorable subjects for the development of both rickets and tetany. Seventy-one hundred and fourteen cases of disease were treated during the years 1896 and 1897, and among them was one case of tetany. One hundred and ninety-six were classified as rickets. This number, however, comprises only those cases in which rachitis was the condition demanding treatment, and does not include the far greater number of cases in which rickets was present as a complication. The one case of tetany, which is reported in full below, showed but few evidences of rickets. It would seem from these figures, therefore, that rachitis is not a factor of great importance in the etiology of tetany. I have, during the last five years, seen six cases of tetany in infants. Three were mild and three severe. The histories of these are as follows:

Case 1.—Annie P., four months old; was seen at the Massachusetts Charitable Eye and Ear Infirmary on account of a suspicious eruption. The mastoid disease, for which an operation had been performed some days before, was progressing favorably. Nothing was known as to her history, and the contractures had not been noted. There was slight diarrhea and the temperature was moderately elevated. Handling was somewhat painful. There were frequent, short, typic paroxysmal contractures of the hands and arms. No laryngospasm; facial phenomena absent; Trousseau's symptom easily elicited; knee jerks exaggerated. There were no evidences of rickets. She unfortunately soon passed out of observation, and nothing is known as to the further progress of the case.

Case 2.—Gladys R., colored, four months old, was brought

to the Infants' Hospital chiefly because she did not gain weight. She had always been feeble, and was said to weigh less than at birth. She had been fed on a varied collection of patent "foods," the mixture at the time being one of Mellins' Food and boiled milk. She had all of this mixture which she would take at any time. There was no vomiting, some abdominal pain, and a moderate diarrhea. She had "spasms" frequently, in which she "wriggled and clenched her hands." No laryngospasm. She was poorly developed, but fairly nourished. There was no evidence of rickets. During the examination there were frequent short, intermittent, typic contractures in the arms and hands. There was apparently no pain, either on handling or during the paroxysms. The facial phenomenon could not be obtained, but Trousseau's symptom was easily elicited. She was given a carefully modified milk, but no drugs. When seen, eight days later, she had gained ten ounces, and the gastro intestinal symptoms had diminished greatly. There had been no spasms for some days. Trousseau's symptom, however, was easily developed. She was then lost sight of, and nothing is known as to the final outcome of the case.

Case 3.—Rebecca B., ten months old, was brought to the Infants' Hospital, May 6. Since the first of January she had had one or more "spasms" a day, lasting from a few seconds to five minutes. They also sometimes occurred during sleep. In these "spasms" the upper part of the body stiffened, the arms were flexed at the elbows, the thumb contracted into the palm, and she "got black in the face." For the same length of time she had had frequent attacks of noisy breathing. An older child had had a similar trouble, lasting a month at one time. She was fed on breast milk and on a mixture of cow's milk and water. She seldom vomited, and the dejections were normal, except for an occasional curd. Her surroundings were very good for a child of the poorer classes. She was well developed and nourished. Color good. Skin normal. Anterior fontanelle, six c.c. by eight c.c. No craniotabes. Two teeth. Spine normal. Heart and lungs normal. A considerable rosary. Flaring of lower chest. Abdomen large. Spleen palpable. Epiphyses at wrists enlarged. During the examination she had a typic contracture, involving the arms only, lasting about half a minute. She also had several attacks of laryngospasm. The facial phenomenon was easily elicited, but Trousseau's symptom could not be produced. The reflexes were normal. She was put on a carefully modified milk in addition to the breast, and given small doses of bromid. The spasms ceased on the 10th and the laryngismus on the 23d. The facial phenomenon was present on the 10th, but had disappeared on the 13th. No new symptoms developed.

Case 4.—James C., six months old, was seen in consultation with Dr. J. L. Ames, Jan. 25, 1898. He was a large, strong

baby at birth, but was completely upset by improper feeding during the first six weeks. From that time on his digestion was feeble, although he was carefully and correctly fed. He did not gain normally, and vomited from time to time. The stools, however, were normal. His care and surroundings were irreproachable. On the 19th day he began to vomit again, but his dejections remained normal. He cried much, and seemed to be in pain. His temperature ranged in the neighborhood of 100 degrees. Slight "clenching of the hands,"—first noted on the 20th—gradually increased. He had a mild "spasm" on the 23d. He was cutting several incisor teeth. The urine was diminished in amount. He was fairly developed and nourished. Skin in good condition; rather pale. Fontanelle level—not tense. One tooth; gum distended by another. Slight stomatitis. No rosary. Heart, lungs and abdomen normal. Moderate umbilical hernia. Phimosis. No rigidity of neck, but tendency to hold head backward. Remittent contractures of hands and feet, apparently not attended by pain. Extended thumbs strongly adducted into palms. Little and ring fingers flexed at metacarpo-phalangeal joint, extended at phalangeal; other fingers firmly flexed. Slight flexion at wrists. Elbow and shoulder joints free. Feet in perfectly typic position. Toes sharply flexed at metatarso phalangeal joint, extended at phalangeal. Foot much arched and extended. Knees and hips flexed. Reduction of spasm not painful. Facial phenomenon doubtful. Trousseau's symptom easily elicited. Knee-jerks lively.

He was given a different modification of milk, calomel, salol and bromid. The further treatment was on these same lines. He had numerous spasms during the next few days and the paroxysmal contractures increased in severity. Under careful feeding all the symptoms of indigestion disappeared during the next month, but there was no gain in weight. After the first week the contractures diminished rapidly in frequency and severity and were entirely gone in a month.

Case 5.—John W., four months old, was brought to the City Hospital, Oct. 21, 1897. There was no specific history, and the other children were in every way normal. He was born at full term, after a rapid, normal, vertex labor. Convulsions are said to have begun on the day of his birth, and to have continued until some time in July. The mother also states that she noticed contraction of the fingers after the first convulsion, and that it has persisted since. I saw him at the Infants' Hospital in July, however, but noticed no contractures. I sent him to the Summer Hospital at Rainsford Island, with the diagnosis of "starvation." He remained there for some weeks, but no contractures were noted.

He was fed on condensed milk previous to his admission to Rainsford, and on modified milk during his stay there. He was then given Mellins' Food, which he was taking when seen.

12

He had been vomiting for some weeks. The bowels moved two or three times a day, the dejections being large, yellowish-white and undigested. He slept but little and cried constantly. "The whole body was constantly rigid, the spasm not even relaxing in sleep." Motion was painful.

JOHN W.

Poorly developed and emaciated; skin dry; fontanelle level; snuffles; no teeth; mouth normal; tendency to laryngospasm when crying; unable to hold up head when sitting; when lying, head held backward stiffly, but spasm can be overcome; facial phenomenon not obtained; heart and lungs normal; no

rosary; abdomen large and tense; slight umbilical hernia; no enlargement of liver or spleen; phimosis; no glandular enlargement; arms held rigidly flexed at the elbow; shoulders also somewhat rigid; wrists slightly flexed, hands in pronation; extended thumbs very strongly adducted across the palms; fingers flexed at metacarpo phalangeal and first phalangeal joints, extended at the second. This spasm varied in intensity, but was never voluntarily relaxed. It could be almost entirely overcome, but only with the causation of considerable pain. Trousseau's symptom was not determined because of the constant spasm. There was a tendency to hold the legs crossed, and at times the knees were flexed and the feet extended. The spasm was always very slight, however, and was easily overcome; knee-jerks exaggerated; electric reactions and sensation not determined.

Warm baths and light rubbing were advised. He was put on a properly modified milk and given small doses of bromid of potassium. Improvement in the digestive symptoms was almost immediate and was soon followed by improvement in the nervous symptoms. By November 7 the spasms were intermittent, occurring only at intervals, and the arms and legs were used a little voluntarily. Trousseau's symptom was easily elicited. By November 27 the spasms were very infrequent and of slight intensity and all motions were free. All trace of spasm disappeared some weeks later and Trousseau's symptom could not be elicited. There has been no recurrence of the symptoms.

Case 6.—Sherry M., 10½ months old, was seen at the Infants' Hospital June 1, 1897.

Family history: No tuberculosis nor syphilis; father and mother each 29 years of age and well.

First child—Instrumental; nursed; always well.

Second child—Instrumental; nursed; always well.

Third child—Instrumental; nursed only; well up to 11 months, when had an attack of vomiting and diarrhea and began to stiffen. Vomiting, constipation and wasting continued and stiffness increased and continued up to death at 18 months. Had no food but breast and beef-juice.

Fourth child—Instrumental; nursed only; well up to 7 months, when had a cough and cold followed by vomiting, constipation, wasting and gradually increasing stiffness ending in death at 11 months. Food was breast, cream and orange juice.

Fifth child—The patient. Normal vertex labor; was well up to 7 months, when it was noted that she was not as active as she had been. Vomiting began and increased, also constipation, which grew steadily worse, the bowels moving only with drugs. At 9½ months she began to hold her head backward, and for the last three was in much the same condition as when first seen. She had the breast only until her sickness began,

14

but after that was given in addition oatmeal gruel, milk and beef-juice and during the last few days a modified milk. She slept but little, and cried almost constantly. All motion seemed to cause pain and increased and brought on spasm. The spasm was never entirely relaxed even during sleep. Fairly developed and nourished. Skin smooth and soft. Color fair; fontanelle normal size; heart, lungs and abdomen normal; no rosary; no glandular enlargement; the arms and legs were in a state of almost constant contracture, which never entirely relaxed. Any motion or excitement caused an increase of the spasm with crying. During the more intense spasms the head was much retracted and the back in the position of moderate opisthotonos. Respiratory spasm was marked, the chest being fixed in the position of full inspiration. The arms were rigid, their position, however, varying in different attacks. The thumbs were always strongly adducted in full extension. The fingers were flexed at the metacarpo phalan-

geal and first phalangeal joints and extended at the second; sometimes extended at the first phalangeal joint also. The legs were held rigidly, the thighs being crossed, the knees extended, the feet slightly extended and the toes flexed. The facial phenomenon was not elicited; Trousseau's symptom was marked; the knee-jerks were normal. The electric reactions and sensation were not examined.

As the history of the other children suggested some trouble with the breast milk, and as this child did better on a mixture of modified milk and breast-milk than on breast-milk alone, the nursing was stopped and she was given modified milk only. She was also given small doses of bromid of potassium. There was no improvement in the spasm, vomiting and constipation. There was also a progressive loss of weight and a continuous temperature, ranging from 99 to 100 degrees. She was therefore sent to the Summer Hospital at Rainsford Island on June 14 and remained there for more than a month. While there she vomited constantly and lost weight steadily. The dejections, however, were normal. Her temperature ranged from

99 to 101 degrees. The contractures continued undiminished. During the next three weeks she improved in every way. She cried less, slept all night and had spasms only when disturbed. The symptoms increased again, however, and she was lost sight of until November 22. During this time she was kept on a modified milk of very low percentages, ordered when she was last seen. She had emaciated to a skeleton, and was very weak. She was vomiting a little. The bowels were constipated but the dejections were normal in character. She slept well and relaxed entirely during sleep. She was quiet when awake but had more or less spasm most of the time. As she was then sixteen months old she was given starchy foods in addition to milk. Passive motion and light rubbing were also advised. Improvement from this time on was slow but progressive. The vomiting ceased but the bowels remained somewhat costive. Gain in weight and strength was steady. The spasm diminished slowly, remaining longest in the hands. It left them about March 1, 1898.

TREATMENT.

It is evident from the obscurity which still exists with regard to the origin and nature of tetany that there can be no specific treatment. As what evidence there is, however, all seems to show that the disease is due to toxic poisoning of some sort, treatment may be directed to the prevention of the formation of toxic substances, and to their elimination, if already formed. It is probable that many of these toxins are absorbed from the alimentary tract and some from the respiratory tract. Others may be produced as the result of abnormal metabolism. The production of alimentary toxins can be best prevented by a suitable diet. Regulation of the diet is probably, therefore, the most important method of preventive treatment. Intestinal antiseptics may also be of use, and are, at any rate, worthy of a trial. The formation and absorption of respiratory toxins can be best prevented by a liberal supply of fresh air and sunlight. Anything which tends to improve the hygienic surroundings is of use. Elimination must naturally take place largely through the intestinal tract, and to a less extent through the kidneys and skin. Emetics, purgatives and lavage of the stomach and colon tend, not only to favor elimination, but also to prevent the absorption

of toxic substances already formed. The kidneys must be kept active by a liberal supply of water and possibly by mild diuretics. The skin may be stimulated by proper hygienic measures.

Symptomatic treatment is also important and consists largely in the avoidance of excitants of spasm and in the employment of antispasmodics. Excitants to be avoided are cold, handling, noise and excitement. Antispasmodic treatment may be local, by the use of such measures as warmth, warm baths, inunctions of oil and light massage, or general, by drugs. The bromids and chloral are the most useful, but opium, belladonna, valerian and musk may be tried. During convulsions chloroform or ether may be administered by inhalation.

LITERATURE.

Blondeau: Quoted by Oddo.
Bonome and Cervesato: La Pediatria, 1895, No. 5 and 6; Revue des Mal. de l'Enfance, Feb., 1896.
Bouchut: Leç. recueilli par Juhel-Rénoy. Paris Méd. 1879, No. 50.
Carpenter: Journal American Medical Association, 1894, xxiii, p. 182.
Cassell: Deutsche Med. Woch., 1897, No. 5.
Chvostek: Wien. Med. Presse, 1876.
Comby: La Médecine Infantile, 1894, No. 4.
Crandall: Archives of Pedriatics, 1895, xii, 920.
Erb: Archiv. f. Phys., iv, 271.
Escherich: Wien. klin. Woch., 1890, No. 40; Berlin klin. Woch., 1897, xxxiv, 861; Traité des Maladies de l'Enfance, Vol. iv, Paris, 1898.
Fischl: Therapeutische Woch., 1896, iii, 973.
Frankl-Hochwart: Die Tetanie, Berlin, 1891; Die Tetanie, Spec. Path. u. Therap., Nothnagel, Wien., 1897, xi, t 2, ab. 4, 79-207.
Griffith: Am. Journ. Med. Sciences, 1895, cix, 158.
Grisolle: Quoted by Oddo.
Hauser: Berlin klin. Woch., 1896, xxxiii, 661, 782.
Heubner: Deutsche Med. Woch., 1896, No. 29.
Kalischer: Revue des Mal. de l'Enfance. 1893, xi, 137; Jahrb. f. Kinderheilk., 1896, xlii, 387.
Kassowitz: Wien. Med. Woch., 1893, No. 13; Wien. Med. Presse, 1897, No. 5.
Krauss: N. Y. Med. Record, 1896, xlix 44.
Lewi: Archives of Pediatrics, 1895, xii, 602.
Loos: Die Tetanie d. Kind. und ihre Berichtungen zur Laryngospasmus, Leipzig, 1892.
Miles: Med. News, 1896, lxviii, 36.
Niemayer: Quoted by Oddo.
Oddo: Révue de Médecine, 1896, xvi, 458, 572, 667, 749.
Orthen: Zeitschr. f. Hyg., 1896, xxii, 1.
Preston: N. Y. Med. Journal, 1895, lxi, 718.
Rehn: Zeitschr. f. Hyg., 1896, xxii, 12.
Romme: Gazette Hebdomadaire de Méd., 1897, xliv, 73.
Sarbo: Deutsche Zeitschr. f. Nervenheilk., 1896, viii.
Schultze: Deutsche Med. Woch., 1882, No. 20.
Stasse: Quoted by Oddo.
Tonnellé: Gazette Médicale, 1832, iii, No. 1.
Tordeus: Jour. de Clin. et de Therap. Infantile, 1897, v, 784.
Trousseau: Cliniques de l'Hotel-Dieu, ii. 207.
Weiss: Samml. Klin. Vorträge, 1881, No. 189.

strated the heightened excitability of the sensory nerves, the slightest pressure on which may cause paræsthesia in the region of distribution.

Diagnosis.—The disease is readily recognized. It is a mistake to call instances of carpo-pedal spasm of children true tetany. It is common to find in rickety children or in cases of severe gastro-intestinal catarrh a transient spasm of the fingers or even of the arms. By many authors these are considered cases of mild tetany, and there are all grades in rickety children between the simple carpo-pedal spasm and the condition in which the four extremities are involved; but it is well, I think, to limit the term *tetany* to the severer affection.

With true tetanus the disease is scarcely ever confounded, as the commencement of the spasm in the extremities, the attitude of the hands, and the etiological factors are very different. Hysterical contractures are usually unilateral.

Treatment.—In the case of children the condition with which the tetany is associated should be treated. Baths and cold sponging are recommended and often relieve the spasm as promptly as in child-crowing. Bromide of potassium may be tried. In severe cases chloroform inhalations may be given. Massage, electricity, and the spinal ice-bag have also been used with success. Cases, however, may resist all treatment, and the spasms recur for many years. The thyroid extract should be tried. Gottstein reports relief in a case of long standing, and Bramwell reports one case of operative tetany and one of the idiopathic form successfully treated in this way.

XI. HYSTERIA.

Definition.—A state in which ideas control the body and produce morbid changes in its functions (Möbius).

Etiology.—The affection is most common in women, and usually appears first about the time of puberty, but the manifestations may continue until the menopause, or even until old age. Men, however, are by no means exempt, and of late years hysteria in the male has attracted much attention. It occurs in all races, but is much more prevalent, particularly in its severer forms, in members of the Latin race. In this country the milder grades are common, but the graver forms are rare in comparison with the frequency with which they are seen in France.

Children under twelve years of age are not very often affected, but the disease may be well marked as early as the fifth or sixth year. One of the saddest chapters in the history of human deception, that of the Salem witches, might be headed *hysteria in children*, since the tragedy resulted directly from the hysterical pranks of girls under twelve years of age.

Of predisposing causes, two are important—heredity and education. The former acts by endowing the child with a mobile, abnormally sensitive nervous organization. We see cases most frequently in families with marked neuropathic tendencies, the members of which have suffered from neuroses of various sorts. Education at home too often fails to inculcate

habits of self-control. A child grows to girlhood with an entirely erroneous idea of her relations to others, and accustomed to have every whim gratified and abundant sympathy lavished on every woe, however trifling, she reaches womanhood with a moral organization unfitted to withstand the cares and worries of every-day life. At school, between the ages of twelve and fifteen, the most important period in her life, when the vital energies are absorbed in the rapid development of the body, she is often cramming for examinations and cooped in close school-rooms for six or eight hours daily. The result too frequently is an active, bright mind in an enfeebled body, ill adapted to subserve the functions for which it was framed, easily disordered, and prone to react abnormally to the ordinary stimuli of life. Among the more direct influences are emotions of various kinds, fright occasionally, more frequently love affairs, grief, and domestic worries. Physical causes less often bring on hysterical outbreaks, but they may follow directly upon an injury or develop during the convalescence from an acute illness or be associated with disease of the generative organs. The name *hysteria* indicates how important was believed to be the part played by the uterus in the causation of the disease. Opinions differ a good deal on this question, but undoubtedly in many cases there are ovarian and uterine disorders the rectification of which sometimes cures the disease. Sexual excess, particularly masturbation, is an important factor, both in girls and boys.

Symptoms.—A useful division is into the convulsive and non-convulsive varieties.

Convulsive Hysteria.—(*a*) *Minor Forms.*—The attack most commonly follows emotional disturbance. It may set in suddenly or be preceded by symptoms, called by the laity "hysterical," such as laughing and crying alternately, or a sensation of constriction in the neck, or of a ball rising in the throat—the *globus hystericus*. Sometimes, preceding the convulsive movements, there may be painful sensations arising from the pelvic, abdominal, or thoracic regions. From the description these sensations resemble auræ. They become more intense with the rising sensation of choking in the neck and difficulty in getting breath, and the patient falls into a more or less violent convulsion. It will be noticed that the fall is not sudden, as in epilepsy, but the subject goes down, as a rule, easily, often picking a soft spot, like a sofa or an easy-chair, and in the movements apparently exercises care to do herself no injury. Yet at the same time she appears to be quite unconscious. The movements are clonic and disorderly, consisting of to-and-fro motions of the trunk or pelvic muscles, while the head and arms are thrown about in an irregular manner. The paroxysm after a few minutes slowly subsides, then the patient becomes emotional, and gradually regains consciousness. When questioned the patient may confess to having some knowledge of the events which have taken place, but, as a rule, has no accurate recollection. During the attack the abdomen may be much distended with flatus, and subsequently a large amount of clear urine may be passed. These attacks vary greatly in character. There may be scarcely any movements of the limbs, but after a nerve storm the patient sinks into a torpid, semi-unconscious condition,

from which she is roused with great difficulty. In some cases from this state the patient passes into a condition of catalepsy.

(*b*) *Major Forms; Hystero-epilepsy.*—This condition has been especially studied by Charcot and his pupils. Typical instances passing through the various phases are very rare in this country. The attack is initiated by certain prodromata, chiefly minor hysterical manifestations, either foolish or unseemly behavior, excitement, sometimes dyspeptic symptoms with tympanites, or frequent micturition. Areas of hyperæsthesia may at this time be marked, the so-called hysterogenic spots so elaborately described by Richet. These are usually symmetrical and situated over the upper dorsal vertebra, and in front in a series of symmetrically placed spots on the chest and abdomen, the most marked being those in the inguinal regions over the ovaries. Painful sensations or a feeling of oppression and a *globus* rising in the throat may be complained of prior to the onset of the convulsion, which, according to French writers, has four distinct stages: (1) Epileptoid condition, which closely simulates a true epileptic attack with tonic spasm (often leading to opisthotonos), grinding of the teeth, congestion of the face, followed by clonic convulsions, gradual relaxation, and coma. This attack lasts rather longer than a true epileptic attack. (2) Succeeding this is the period which Charcot has termed *clownism*, in which there is an emotional display and a remarkable series of contortions or of cataleptic poses. (3) Then in typical cases there is a stage in which the patient assumes certain attitudes expressive of the various passions—ecstasy, fear, beatitude, or erotism. (4) Finally consciousness returns and the patient enters upon a stage in which she may display very varied symptoms, chiefly manifestations of a delirium with the most extraordinary hallucinations. Visions are seen, voices heard, and conversations held with imaginary persons. In this stage patients will relate with the utmost solemnity imaginary events, and make extraordinary and serious charges against individuals. This sometimes gives a grave aspect to these seizures, for not only will the patient at this stage make and believe the statements, but when recovery is complete the hallucination sometimes persists. We seldom see in this country attacks having this orderly sequence. Much more commonly the convulsions succeed each other at intervals for several days in succession. Here is a striking difference between hystero-epilepsy and true epilepsy. In the latter the status epilepticus, if persistent, is always serious, associated with fever, and frequently fatal, while in hystero-epilepsy attacks may recur for days without special danger to life. After an attack of hystero-epilepsy the patient may sink into a state of trance or lethargy, in which she may remain for days.

Non-convulsive Forms.—So complex and varied is the clinical picture of hysteria that various manifestations are best considered according to the systems which are involved.

(1) **Disorders of Motion.**—(*a*) *Paralyses.*—These may be hemiplegic, paraplegic, or monoplegic. Hysterical diplegia is extremely rare. The paralysis either sets in abruptly or gradually, and may take weeks to attain its full development. *There is no type or form of organic paralysis which*

may not be simulated in hysteria. According to Weir Mitchell, the hemiplegias are most frequent in the ratio of four on the left to one on the right side. The face is not affected; the neck may be involved, but the leg suffers most. Sensation is either lessened or lost on the affected side. The hysterical paraplegia is more common than hemiplegia. The loss of power is not absolute; the legs can usually be moved, but do not support the patient. The reflexes may be increased, though the knee-jerk is often normal. A spurious ankle clonus may sometimes be present. The feet are usually extended and turned inward in the equino-varus position. The muscles do not waste and the electrical reactions are normal. Other manifestations, such as paralysis of the bladder or aphonia, are usually associated with the hysterical paraplegia. Hysterical monoplegias may be facial, crural, or brachial. A condition of ataxia sometimes occurs with paresis. The incoördination may be a marked feature, and there are usually sensory manifestations.

(*b*) *Contractures and Spasms.*—An extraordinary variety of spasmodic affections occurs in hysteria, of which the most common are the following: The hysterical contractures may attack almost any group of voluntary muscles and be of the hemiplegic, paraplegic, or monoplegic type. They may come on suddenly or slowly, persist for months or years, and disappear rapidly. The contracture is most commonly seen in the arm, which is flexed at the elbow and wrist, while the fingers tightly grasp the thumb in the palm of the hand; more rarely the terminal phalanges are hyperextended as in athetosis. It may occur in one or in both legs, more commonly the former. The ankle clonus is present; the foot is inverted and the toes are strongly flexed. These cases may be mistaken for lateral sclerosis and the difficulty in diagnosis may really be very great. The spastic gait is very typical, and with the exaggerated knee-jerk and ankle clonus the picture may be characteristic. In 1879 I frequently showed such a case at the Montreal General Hospital as a typical example of lateral sclerosis. The condition persisted for more than eighteen months and then disappeared completely. Other forms of contracture may be in the muscles of the hip, shoulder, or neck; more rarely in those of the jaws—hysterical trismus—or in the tongue. Remarkable indeed are the local contractures in the diaphragm and abdominal muscles, producing a phantom tumor, in which just below and in the neighborhood of the umbilicus is a firm, apparently solid growth. According to Gowers, this is produced by relaxation of the recti and a spasmodic contraction of the diaphragm, together with inflation of the intestines with gas and an arching forward of the vertebral column. They are apt to occur in middle-aged women about the menopause, and are frequently associated with the symptoms of spurious pregnancy—*pseudo-cyesis.* The resemblance to a tumor may be striking, and I have known skilful diagnosticians to be deceived. The only safeguard is to be found in complete anæsthesia, when the tumor entirely disappears. Some years ago I went by chance into the operating-room of a hospital and found a patient on the table under chloroform and the surgeon prepared to perform ovariotomy. The tumor, however, had completely disappeared with full anæsthesia. Mitchell has reported an instance

of a phantom tumor in the left pectoral region just above the breast, which was tender, hard, and dense.

Clonic spasms are more common in hysteria in this country than contractures. The following are the important forms: *Rhythmic hysterical spasm.* This, unfortunately, is sometimes known as rhythmic chorea or hysterical chorea. The movements may be of the arm, either flexion and extension, or, more rarely, pronation and supination. Clonic contractions of the sterno-cleido-mastoid or of the muscles of the jaws or of the rotatory muscles of the head may produce rhythmic movements of these parts. The spasm may be in one or both psoas muscles, lifting the leg in a rhythmic manner eight or ten times in a minute. In other instances the muscles of the trunk are affected, and every few moments there is a bowing movement—salaam convulsions—or the muscles of the back may contract, causing strong arching of the vertebral column and retraction of the head. These movements may often alternate, as in a case in my wards, in which the patient on fine days had regular salaam convulsions, while on wet days the rhythmic spasm was in the muscles of the back and neck. Mitchell has described a rotatory spasm in which the patient rotated involuntarily, usually to the left. More unusual cases are those in which the contractions closely simulate paramyoclonus multiplex. Hysterical athetosis is a rare form of spasm. *Tremor* may be a purely hysterical manifestation, occurring either alone or with paralysis and contracture. It most commonly involves the hands and arms; more rarely the head and legs. The movements are small and quick. In the type *Rendu* the tremor may or may not persist during repose, but it is increased or provoked by volitional movements. Volitional or intentional tremor may exist, simulating closely the movements of insular sclerosis. Buzzard states that many instances of this disease in young girls are mistaken for hysteria.

(2) **Disorders of Sensation.**—*Anæsthesia* is most common, and usually confined to one half of the body. It may not be noticed by the patient. Usually it is accurately limited to the middle line and involves the mucous surfaces and deeper parts. The conjunctiva, however, is often spared. There may be hemianopia. This symptom may come on slowly or follow a convulsive attack. Sometimes the various sensations are dissociated and the anæsthesia may be only to pain and to touch. The skin of the affected side is usually pale and cool, and a pin-prick may not be followed by blood. With the loss of feeling there may be loss of muscular power. Curious trophic changes may be present, as in an interesting case of Weir Mitchell's, in which there was unilateral swelling of the hemiplegic side.

A phenomenon to which much attention has been paid is that of transference. By metallotherapy, the application of certain metals, the anæsthesia or analgesia can be transferred to the other side of the body. It has been shown, however, that this phenomenon may be caused by the electro-magnet and by wood and various other agents, and is probably entirely a mental effect. The subject has no practical importance, but it remains an interesting and instructive chapter in Gallic medical history.

Hyperæsthesia.—Increased sensitiveness and pains occur in various parts of the body. One of the most frequent complaints is of pain in the head,

usually over the sagittal suture, less frequently in the occiput. This is described as agonizing, and is compared to the driving of a nail into the part; hence the name *clavus hystericus*. Neuralgias are common. Hyperæsthetic areas, the hysterogenic points, exist on the skin of the thorax and abdomen, pressure upon which may cause minor manifestations or even a convulsive attack. Increased sensitiveness exists in the ovarian region, but is not peculiar to hysteria. Pain in the back is an almost constant complaint of hysterical patients. The sensitiveness may be limited to certain spinous processes, or it may be diffuse. In hysterical women the pains in the abdomen may simulate those of gastralgia and of gastric ulcer, or the condition may be almost identical with that of peritonitis; more rarely the abdominal pains closely resemble those of appendix disease.

Special Senses.—Disturbances of taste and smell are not uncommon and may cause a good deal of distress. Of ocular symptoms, retinal hyperæsthesia is the most common, and the patients always prefer to be in a darkened room. Retraction of the field of vision is common and usually follows a convulsive seizure. It may persist for years. The color perception may be normal even with complete anæsthesia, and in this country the achromatopsia does not seem to be nearly so common an hysterical manifestation as in Europe. Hysterical deafness may be complete and may alternate or come on at the same time with hysterical blindness. Hysterical amaurosis may occur in children. One must carefully distinguish between functional loss of power and simulation.

(3) **Visceral Manifestations.**—*Respiratory Apparatus.*—Of disturbances in the respiratory rhythm, the most frequent, perhaps, is an exaggeration of the deeper breath, which is taken normally every fifth or sixth inspiration, or there may be a "catching" breathing, such as is seen when cold water is poured over a person. Hysterical dyspnœa is readily recognized, as there is no special distress and the pulse is usually normal. I have met with a remarkable case following trauma in which the respirations rose above 130 in the minute. Among laryngeal manifestations aphonia is the most frequent and may persist for months or even years without other special symptoms of the disease. Spasm of the muscles may occur with violent inspiratory efforts and great distress, and may even lead to cyanosis. Hiccough, or sounds resembling it, may be present for weeks or months at a time. Among the most remarkable of the respiratory manifestations are the hysterical cries. These may mimic the sounds produced by animals, such as barking, mewing, or grunting, and in France epidemics of them have been repeatedly observed. Extraordinary cries may be produced, either inspiratory or expiratory. I saw at Wagner's clinic at Leipsic a girl of thirteen or fourteen, who had for many weeks given utterance to a remarkable inspiratory cry somewhat like the whoop of whooping-cough, but so intense that it was heard at a long distance. It was incessant, and the girl was worn to a skeleton. Attacks of gaping, yawning, and sneezing may also occur.

The hysterical cough is a frequent symptom, particularly in young girls. It may occur in paroxysms, but is often a dry, persistent, croaking cough, extremely monotonous and unpleasant to hear. Sir Andrew Clark

Under hysterical dyspnoea may be noted what is known as the syndrome of Briquet, in which there is shortness of breath, suppression of the voice, and no contraction of the diaphragm, apparently due to hysterical paralysis of this organ. The anæsthesia is extreme.

has called attention to a loud, barking cough (*cynobex hebetica*) occurring about the time of puberty, chiefly in boys belonging to neurotic families. The attacks, which last about a minute, recur frequently.

There is a peculiar form of hæmoptysis which may be very deceptive and lead to the diagnosis of pulmonary disorders. Wagner describes the sputum as a pale-red fluid—not so bright in color as in ordinary hæmoptysis; on settling it presents a reddish-brown sediment. It contains particles of food, pavement epithelium, red corpuscles, and micrococci, but no cylindrical or ciliated epithelium. It probably comes from the mouth or pharynx.

Digestive System.—Disturbed or depraved appetite, dyspepsia, and gastric pains are common in hysterical patients. The patient may have difficulty in swallowing the food, apparently from spasm of the gullet. There are instances in which the food seems to be expelled before it reaches the stomach. In other cases there is incessant gagging. In the hysterical vomiting the food is regurgitated without much effort and without nausea. This feature may persist for years without great disturbance of nutrition. The most striking and remarkable digestive disturbance in hysteria is the *ancrexia nervosa* described by Sir William Gull. "To call it loss of appetite—anorexia—but feebly characterizes the symptom. It is rather an annihilation of appetite, so complete that it seems in some cases impossible ever to eat again. Out of it grows an antagonism to food which results at last and in its worst forms in spasm on the approach of food, and this in turn gives rise to some of those remarkable cases of survival for long periods without food" (Mitchell). As this goes on there may be an extreme degree of muscular restlessness, so that the patients wander about until exhausted. Nothing more pitiable is to be seen in practice than an advanced case of this sort. It is usually in a young girl, sometimes as early as the eleventh or twelfth, more commonly between the fifteenth and twentieth years. The emaciation is frightful, and scarcely exceeded by that of cancer of the œsophagus. The patient finally takes to bed, and in extreme cases lies upon one side with the thighs and legs flexed, and contractures may occur. Food is either not taken at all or only upon urgent compulsion. The skin becomes wasted, dry, and covered with bran-like scales. No food may be taken for several weeks at a time, and attempts to feed may be followed by severe spasms. Although the condition looks so alarming, these cases, when removed from their home surroundings and treated by Weir Mitchell's method, sometimes recover in a remarkable way. Death, however, may follow with extreme emaciation. In a fatal case under my care the girl weighed only 49 pounds. No lesions were found post mortem.

Among intestinal symptoms flatulency is one of the most distressing, and is usually associated with the condition of peristaltic unrest (Kussmaul). Frequent discharges of fæces may be due to disturbance in either the small or large bowel. An obstinate form of diarrhœa is found in some hysterical patients, which proves very intractable and is associated especially with the taking of food. It seems an aggravated form of the looseness of bowels to which so many nervous people are subject on emotion or the tendency which some have to diarrhœa immediately after eating.

An entirely different form is that produced by what Mitchell calls the irritable rectum, in which scybala are passed frequently during the day, sometimes with great violence. Constipation is more frequent, however, and may be due to a loss of power in the muscles of the bowel, or in the abdominal muscles. In extreme cases the bowels may not be moved for two or three weeks, leading to great accumulation of fæces. Other disturbances are ano-spasm or intense pain in the rectum apart from any fissure.

Cardio-vascular.—Rapid action of the heart on the slightest emotion, with or without the subjective sensation of palpitation, is often a source of great distress. A slow pulse is less frequent. Pains about the heart may simulate angina, the so-called hysterical or pseudo-angina, which has already been considered. Flushes in various parts are among the most common symptoms. Sweating occasionally occurs.

Among the more remarkable vaso-motor phenomena are the so-called stigmata or hæmorrhages in the skin, such as were present in the celebrated case of Louise Lateau. In many cases these are undoubtedly fraudulent, but if, as appears credible, such bleeding may exist in the hypnotic trance, there seems no reason to doubt its occurrence in the trance of prolonged religious ecstasy.

Joint Affections.—To Sir Benjamin Brodie and Sir James Paget we owe the recognition of these extraordinary manifestations of hysteria. Perhaps no single affection has brought more discredit upon the profession, for the cases are very refractory, and finally fall into the hands of a charlatan or faith-healer, under whose touch the disease may disappear at once. Usually it affects the knee or the hip, and may follow a trifling injury. The joint is usually fixed, sensitive, and swollen. The surface may be cool, but sometimes the local temperature is increased. To the touch it is very sensitive and movement causes great pain. In protracted cases the muscles about the joint are somewhat wasted, and in consequence it looks larger. The pains are often nocturnal, at which time the local temperature may be much increased. While, as a rule, neuromimetic joints yield to proper management, there are interesting instances in the literature in which organic change has succeeded the functional disturbance. In the remarkable case reported in Weir Mitchell's lectures, the hysterical features were pronounced, and, on account of the chronicity, the disease of the knee-joint was considered organic by such an authority as Billroth. Sands found the joint surfaces normal, and the thickening to be due to inflammatory products outside the capsule.

Intermittent hydrarthrosis may be a manifestation of hysteria, occurring in the knee or other joints, sometimes with transient paresis.

Mental Symptoms.—The psychical condition of an hysterical patient is always abnormal, and the disease occupies the ill-defined territory between sanity and insanity. In a large number of cases the patients are really insane, particularly in the perversion witnessed in the moral sphere. Not the slightest dependence can be placed upon their statements, and they will for months or years deceive friends, relatives, and physician. This appears to result partly, but not wholly, from a morbid craving for sympathy. It is really due to an entire unhinging of the moral nature.

Illustrations unmodern see remarkable
case Journal. Nov 9th 1898

Hysterical patients may become insane and display persistent hallucinations and delirium, alternating perhaps with emotional outbursts of an aggravated character. For weeks or months they may be confined to bed, entirely oblivious to their surroundings, with a delirium which may simulate that of delirium tremens, particularly in being associated with loathsome and unpleasant animals. The nutrition may be maintained, but in these cases there is always a very heavy, foul breath. With seclusion and care recovery usually takes place within three or four months. At the onset of these attacks and during convalescence the patients must be incessantly watched, as a suicidal tendency is by no means uncommon. I have been accustomed to speak of this condition as the *status hystericus*.

Of hysterical manifestations in the higher centres that of trance is the most remarkable. This may develop spontaneously without any convulsive seizure, but more frequently, in this country at least, it follows hysteroid attacks. Catalepsy, a condition in which the limbs are plastic and remain in any position in which they are placed, may be present.

The Metabolism in Hysteria.—The studies of Gilles de la Tourette and Cathelineau, under Charcot's direction, have shown that in the ordinary forms of hysteria the urine does not show quantitative or qualitative changes, but in the severer types, characterized by convulsions, etc., there are important modifications: reduction in the urates and phosphates; the ratio of the earthy to the alkaline phosphates, normally 1 : 3, is 1 : 2, or even 1 : 1. The urine is also reduced in amount. They think that these changes might sometimes serve to differentiate convulsive hysteria from epilepsy, in which there is always an increase in the solid constituents after a seizure.

Hysterical Fever.—In hysteria the temperature, as a rule, is normal. The cases with fever may be grouped as follows: (*a*) Instances in which the fever is the sole manifestation. These are rare, but I have seen at least two cases in which the chronic course, the retention of the nutrition, and the entirely negative condition of the organs left no other diagnosis possible. In a case recently under observation the patient has had for four or five years an afternoon rise of temperature, reaching usually to 102° or 103°. She was well nourished and presented no pronounced hysterical symptoms, but there was a marked neurotic history on one side and a form of interrupted sighing respiration so often seen in hysteria.

(*b*) Cases of hysterical fever with spurious local manifestations. These are very troublesome and deceptive cases. The patient may be suddenly taken ill with pain in various regions and elevation of temperature. The case may simulate meningitis. There may be pain in the head, vomiting, contracted pupils, and retraction of the neck—symptoms which may persist for weeks—and some anomalous manifestation during convalescence may alone indicate to the physician that he has had to deal with a case of hysteria, and has not, as he perhaps flattered himself, cured a case of meningitis. Mary Putnam Jacobi, in a recent article on hysterical fever, mentions a case in the service of Cornil which was admitted with dyspnœa, slight cyanosis, and a temperature of 39° C. The condition proved to be hysterical. There is also an hysterical pseudo-phthisis with pain in the

chest, slight fever, and the expectoration of a blood-stained mucus. The cases of hysterical peritonitis may also show fever.

(*c*) *Hysterical Hyperpyrexia.*—It is a suggestive fact that the cases of paradoxical temperatures reported of late years, in which the thermometer has registered 112° to 120° or more, have been in women. Fraud has been practised in some of these, but others have to be accepted, though their explanation is impossible under our known laws. Jacobi has reported a case in which the temperature rose to 148° F. (65.5° C.). The Omaha case, in which the temperature was recorded at 170° F., has, I am informed on good authority, proved a fraud.

Diagnosis.—Inquiry into the occurrence of previous manifestations and the mental conditions may give important information. These questions, as a rule, should not be asked the mother, who of all others is least likely to give satisfactory information about the patient's condition. The occurrence of the globus hystericus, of emotional attacks, of weeping and crying, are always suggestive. The points of difference between the convulsive attacks and true epilepsy were referred to in their description, and as a rule little difficulty is experienced in distinguishing between the two conditions. The hysterical paralyses are very variable and apt to be associated with anæsthesia. The contractures may at times be very deceptive, but the occurrence of areas of anæsthesia, of retraction of the visual field, and the development of minor hysterical manifestations, give valuable indications. The contractures disappear under full anæsthesia. Special care must be taken not to confound the spastic paraplegia of hysteria with lateral sclerosis.

The visceral manifestations are usually recognized without much difficulty. The practitioner has constantly to bear in mind the strong tendency in hysterical patients to practise deception.

Treatment.—The prophylaxis in hysteria may be gathered from the remarks on the relation of education to the disease. The successful treatment of hysteria demands qualities possessed by few physicians. The first element is a due appreciation of the nature of the disease on the part of the physician and friends. It is pitiable to think of the misery which has been inflicted on these unhappy victims by the harsh and unjust treatment which has resulted from false views of the nature of the trouble; on the other hand, worry and ill-health, often the wrecking of mind, body, and estate, are entailed upon the near relatives in the nursing of a protracted case of hysteria. The minor manifestations, attacks of the vapors, the crying and weeping spells, are not of much moment and rarely require treatment. The physical condition should be carefully looked into and the mode of life regulated so as to insure system and order in everything. A congenial occupation offers the best remedy for many of these manifestations. Any functional disturbance should be attended to and a course of tonics prescribed. Special attention should be paid to the action of the bowels.

Valerian and asafœtida are often of service. For the pains in various parts, particularly in the back, the thermo-cautery and static electricity will be found invaluable. Morphia should be withheld. In the convulsive

In what has been called the splanchnic group, characterized by anorexia, etc., death may follow fee
Jos. Fraenkel, Med. News, Jan. 5, 1901.

seizures, particularly in the minor forms, it is often best, after settling the patient comfortably, to leave her. When she comes to, and finds herself alone and without sympathy, the attacks are less likely to be repeated. There is, as a rule, no cure for the hysterical manifestations of women, otherwise in good health, who are, as Mitchell says, "fat and ruddy, with sound organs and good appetites, but ever complain of pains and aches, and ever liable on the least emotional disturbance to exhibit a quaint variety of hysterical phenomena."

To treat hysteria as a physical disorder is, after all, radically wrong. It is essentially a mental and emotional anomaly, and the important element in the treatment is moral control. At home, surrounded by loving relatives who misinterpret entirely the symptoms and have no appreciation of the nature of the disease, the severer forms of hysteria can rarely be cured. The necessary control is impossible; hence the special value of the method introduced by Weir Mitchell, which is particularly applicable to the advanced cases which have become chronic and bedridden. The treatment consists in isolation, rest, diet, massage, and electricity. Separation from friends and sympathetic relatives must be absolute, and can rarely, if ever, be obtained in the individual's home. An essential element in the treatment is an intelligent nurse. No small share of the success which has attended the author of this plan has been due to the fact that he has persistently chosen as his allies bright, intelligent women. The details of the plan are as follows: The patient is confined to bed and not allowed to get up, nor, at first, in aggravated cases, to read, write, or even to feed herself. Massage is used daily, at first for twenty minutes or half an hour, subsequently for a longer period. It is essential as a substitute for exercise. The induction current is applied to the various muscles and to the spine. Its use, however, is not so essential as that of massage. The diet may at first be entirely of milk, 4 ounces every two hours. It is better to give skimmed milk, and it may be diluted with soda water or barley water and, if necessary, peptonized. After a week or ten days the diet may be increased, the amount of milk still being kept up. A chop may be given at midday, a cup of coffee or cocoa with toast or bread and butter or a biscuit with the milk. The patients usually fatten rapidly as the solid food is added, and with the gain there is, as a rule, a diminution or cessation of the nervous symptoms. The milk is the essential element in the diet, and is in itself amply sufficient.

The remarkable results obtained by this method are now universally recognized. The plan is more applicable to the lean than to fat, flabby hysterical patients. Not only is it suitable for the more obstinate varieties of hysteria with bodily manifestations, but in the cases with mental symptoms the seclusion and separation from relatives and friends are particularly advantageous. In the hysterical vomiting Debove's method of forced feeding may be used with benefit. For the innumerable minor manifestations of hysteria and for the simulations the indications for treatment are usually clear. Of late, hypnotism has been extensively used in the treatment of hysteria. Occasionally in cases of hysterical contractions or paralysis it is of benefit, but any one who has seen the development of this method

as practised at present in France must feel that it is a two-edged sword and that the constant repetition in the same patient is fraught with danger. In the cases in which we have tried it here the success has not been marked.

XII. NEURASTHENIA.

Definition.—A condition of weakness or exhaustion of the nervous system, giving rise to various forms of mental and bodily inefficiency.

The term, an old one, but first popularized by Beard, covers an ill-defined, motley group of symptoms, which may be either general and the expression of derangement of the entire system, or local, limited to certain organs; hence the terms cerebral, spinal, cardiac, and gastric neurasthenia.

Etiology.—The causes may be grouped as hereditary and acquired.

(*a*) *Hereditary.*—We do not all start in life with the same amount of nerve capital. Parents who have led irrational lives, indulging in excesses of various kinds, or who have been the subjects of nervous complaints or of mental trouble, may transmit to their children an organization which is defective in what, for want of a better term, we must call "nerve force." Such individuals start handicapped with a neuropathic predisposition, and furnish a considerable proportion of our neurasthenic patients. As van Gieson sonorously puts it, "the potential energies of the higher constellations of their association centres have been squandered by their ancestors."

Besides such forms of hereditary neuropathy, which we have to look upon as instances of injury to the germ-plasm derived from one or both of the parents, there have to be considered those cases in which during intra-uterine life there have been conditions which interfered with the proper development and nutrition of the embryo. So long as these individuals are content to transact a moderate business with their life capital, all may go well, but there is no reserve, and in the exigencies of modern life these small capitalists go under and come to us as bankrupts.

(*b*) *Acquired.*—The functions, though perverted most readily in persons who have inherited a feeble organization, may also be damaged in persons with no neuropathic predisposition by exercise which is excessive in proportion to the strength—i. e., by strain. The cares and anxieties attendant upon the gaining of a livelihood may be borne without distress, but in many persons the strain becomes excessive and is first manifested as *worry*. The individual loses the distinction between essentials and non-essentials, trifles cause annoyance, and the entire organism reacts with unnecessary readiness to slight stimuli, and is in a state which the older writers called irritable weakness. If such a condition be taken early and the patient given rest, the balance is quickly restored. In this group may be placed a large proportion of the neurasthenics which we see in this country, particularly among business men, teachers, and journalists. Neurasthenia may follow the infectious diseases, particularly influenza, typhoid fever, and syphilis. The abuse of certain drugs, alcohol, tobacco, morphine may lead to a high grade of neurasthenia, though the drug habit is more often a result rather than a cause of the neurasthenia. Other causes more subtle, yet potent, and

less easily dealt with, are the worries attendant upon love affairs, religious doubts, and the sexual passion. Sexual excesses have undoubtedly been exaggerated as a cause of neurasthenia, but that they are responsible in a number of instances is certain.

The traumatic forms, especially those following upon railway accidents, will be separately considered.

Symptoms.—These are extremely varied, and may be general or localized; more often a combination of both. The appearance of the patient is suggestive, sometimes characteristic, but difficult to describe. Important information can be gained by the physician if he observe the patient closely as he enters the room—the way he is clothed, the manner in which he holds his body, his facial expression, and the humor which he is in. Loss of weight and slight anæmia may be present. The physical debility may reach a high grade and the patient may be confined to bed. Mentally the patients are usually low-spirited and despondent, in women frequently emotional.

The local symptoms may dominate the situation, and there have accordingly been described a whole series of types of the disease—cerebral, spinal, cardio-vascular, gastric, and sexual. In all forms there is a striking lack of accordance between the symptoms of which the patient complains and the objective changes discoverable by the physician. In nearly every clinical type of the disease the predominant symptoms are referable to pathological sensations and the psychic effects of these. Imperfect sleep is also complained of by a majority of patients, or, if not complained of, is found to exist on inquiry.

In the cerebral or psychic form the symptoms are chiefly connected with an inability to perform the ordinary mental work. Thus a row of figures cannot be correctly added, the dictation or the writing of a few letters is a source of the greatest worry, the transaction of petty details in business is a painful effort, and there is loss of power of fixed attention. With this condition there may be no headache, the appetite may be good, and the patient may sleep well. As a rule, however, there are sensations of fulness and weight or flushes, if not actual headache. Sleeplessness is a frequent concomitant of the cerebral form, and may be the first manifestation. Some of these patients are good-tempered and cheerful, but a majority are moody, irritable, and depressed.

Hyperæsthesia, especially to sensations of pain, is one of the main characteristics of almost all neurasthenic individuals. The sensations are nearly always referred to some special region of the body—the skin, eye muscles, the joints, the blood-vessels, or the viscera. It is frequently possible to localize a number of points painful to pressure (Valleix's points). In some patients there is marked vertigo, occasionally even resembling that of Ménière's disease.

If such pathological sensations continue for a long time the mood and character of the patient gradually alter. The so-called "irritable humor" develops. Many obnoxiously egoistic individuals met with in daily life are in reality examples of psychic neurasthenia. Everything is complained of. The individual demands the greatest consideration for his condition; feels

that he has been deeply insulted if his desires are not always immediately granted. He may at the same time have but little consideration for others. Indeed, in the severer forms of the disease he may show a malicious pleasure in attempting to make people who seem happier than himself uncomfortable. Such patients complain frequently that they are "misunderstood" by their fellows.

In many cases the so-called "anxiety conditions" gradually develop; one scarcely ever sees a case of advanced neurasthenia without the existence of some form of "anxiety." In the simpler forms of anxiety (nosophobic) there may be only a fear of impending insanity or of approaching death or of apoplexy. More frequently the anxious feeling is localized somewhere in the body—in the præcordial region, in the head, in the abdomen, in the thorax, or more rarely in the extremities.

In some cases the anxiety becomes intense and the patients are restless, and declare that they do not know what to do with themselves. They may throw themselves upon a bed, crying and complaining, and making convulsive movements with the hands and feet. Suicidal tendencies are not uncommon in such cases, and patients may in desperation actually take their own lives.

Involuntary mental activity may be very troublesome; the patient complains that when he is overtired thoughts which he cannot stop or control run through his head with lightning-like rapidity. In other cases there is marked absence of mind, the individual's mind being so filled up owing to the overexcitability of latent memory pictures that he is unable to form the proper associations for ideas called up by external stimuli. Sometimes a patient complains that a definite word, a name, a number, a melody, or a song keeps running in his head in spite of all he can do to abolish it.

In the severer cases of psychic neurasthenia the so-called "phobias" are common. The most frequent form perhaps is *agoraphobia*, in which patients the moment they come into an open space are oppressed by an exaggerated feeling of anxiety. They seem "frightened to death," and commence to tremble all over; they complain of compression of the thorax and palpitation of the heart. They may break into profuse perspiration and assert that they feel as though chained to the ground or that they cannot move a step. It is remarkable that in some such cases the open space can be crossed if the individual be accompanied by some one, even by a child, or if he carry a stick or an umbrella! Other people are afraid to be left alone (monophobia), especially in a closed compartment (claustrophobia).

The fear of people and of society is known as anthropophobia. A whole series of other phobias have been described—batophobia, or the fear that high things will fall; pathophobia, or fear of disease; siderodromophobia, or fear of a railway journey; siderophobia or astrophobia, fear of thunder and lightning. Occasionally we meet with individuals who are afraid of everything and every one—victims of the so-called pantophobia.

The *special senses* may be disturbed, particularly vision. An aching or weariness of the eyeballs after reading a few minutes or flashes of light are common symptoms. The "irritable eye," the so-called nervous or neurasthenic asthenopia, is familiar to every family physician. According to

Binswanger, the essence of the asthenopic disturbance consists in pathological sensations of fatigue in the ciliary muscles or the medial recti.

There may be acoustic disturbances—hyperalgesia and even true hyperacusia.

One of the most common of all the symptoms of neurasthenia is the *pressure in the head* complained of by these patients. This symptom, variously described, may be diffuse, but is more frequently referred to some one region—frontal, temporal, parietal, or occipital.*

When the *spinal symptoms* predominate—spinal irritation or spinal neurasthenia—in addition to many of the features just mentioned, the patients complain of weariness on the least exertion, of weakness, pain in the back, intercostal neuralgiform pains, and of aching pains in the legs. There may be spots of local tenderness on the spine. The rachialgia may be spontaneous, or may be noticed only on pressure or movement. Occasionally there may be disturbances of sensation, particularly a feeling of numbness and tingling, and the reflexes may be increased. Visceral neuralgias, especially in connection with the genital organs, are frequently met with. The aching pain in the back or in the back of the neck is the most constant complaint in these cases. In women it is often impossible to say whether this condition is one of neurasthenia or hysteria. It is in these cases that the disturbances of muscular activity are most pronounced, and in the French writings *amyosthenia* particularly plays an important *rôle*. The symptoms may be irritative or paretic, or a combination of both. Disturbances of coördination are not uncommon in the severer forms. These are particularly prone to involve the associated movements of the eye muscles leading to asthenopic lack of accommodation. Drooping of one eyelid is very common, probably owing to insufficient innervation on the part of the sympathetic rather than to paresis of the nervus oculomotorius. Occasionally Romberg's symptom may be present, and the patient, or even his physician, may fear a beginning tabes. More rarely there is disturbance of such finely coördinated acts as writing and articulation, not unlike those seen at the onset of general paresis. Such symptoms are always alarming, and the greatest care must be taken in establishing a diagnosis. That they may be the symptoms of pure neurasthenia, however, can no longer be doubted.

The reflexes in neurasthenia are usually increased, the deep reflexes especially never being absent. The condition of the superficial reflexes is less constant, though these, too, are usually increased. The pupils are often dilated, and the reflexes are usually normal. There may be inequality of the pupils in neurasthenia, a point which Pelizaeus has especially emphasized.

In another type of cases the muscular weakness is extreme, and may go on even to complete motor helplessness. Very thorough examination is necessary before deciding as to the nature of the affection, since in some

* For an exhaustive consideration of the mental symptoms of neurasthenia, see the Shattuck Lecture, by Cowles (Boston Medical and Surgical Journal, 1891), as well as two German monographs, that of Binswanger (1896), and that of Löwenfeld. The French treatise of Bouveret (1891) is also valuable. F. C. Müller's Handbuch der Neurasthenie (Leipzig, 1893) contains an excellent bibliography of this subject.

instances serious mistakes have been made. Here belong the *atremia* of Neftel, the *akinesia algera* of Möbius, and the neurasthenic form of *astasia abasia* described by Binswanger.

In other cases the *cardio-vascular* symptoms are the most distressing, and may occur with only slight disturbance of the cerebro-spinal functions, though the conditions are nearly always combined. Palpitation of the heart, irregular and very rapid action (neurasthenic tachycardia), and pains and oppressive feelings in the cardiac region are the most common symptoms. The slightest excitement may be followed by increased action of the heart, sometimes associated with sensations of dizziness and anxiety, and the patients frequently have the idea that they suffer from serious disease of this organ. Attacks of pseudo-angina may occur.

Vaso-motor disturbances constitute a special feature of many cases. Flushes of heat, especially in the head, and transient hyperæmia of the skin may be very distressing symptoms. Profuse sweating may occur, either local or general, and sometimes nocturnal. The pulse may show interesting features, owing to the extreme relaxation of the peripheral arterioles. The arterial throbbing may be everywhere visible, almost as much as in aortic insufficiency. The pulse, too, may under these circumstances have a somewhat water-hammer quality. The capillary pulse may be seen in the nails, on the lips, or on the margins of a line drawn upon the forehead, and I have on several occasions seen pulsation in the veins of the back of the hand. A characteristic symptom in some cases is the *throbbing aorta*. This "preternatural pulsation in the epigastrium," as Allan Burns calls it, may be extremely forcible and suggest the existence of abdominal aneurism. The subjective sensations associated with it may be very unpleasant, particularly when the stomach is empty.

In women especially, and sometimes in men, the peripheral blood-vessels are contracted, the extremities are cold, the nose is red or blue, and the face has a pinched expression. These patients feel much more comfortable when the cutaneous vessels are distended, and resort to various means to favor this (wearing of heavy clothing, use of diffusible stimulants).

The general features of *gastro-intestinal neurasthenia* have been dealt with under the section of nervous dyspepsia. The connection of these cases with dilatation of the stomach, floating kidney, and the condition which Glénard calls *enteroptosis* has already been mentioned.

Sexual neurasthenia is a condition in which there is an irritable weakness of the sexual organs manifested by nocturnal emissions, unusual depression after intercourse, and often by a distressing dread of impotence. The mental condition of these patients is most pitiable, and they fall an easy prey to quacks and charlatans of all kinds.

Spermatorrhœa is the bugbear of the majority. They complain of continued losses, usually without accompanying pleasurable sensations. After defecation or micturition there may be seminal discharges. Microscopic examination sometimes reveals the presence of spermatozoa. Actual nervous impotence is not uncommon. The "painful testicle" is a well-known neurasthenic phenomenon.

In the severer cases, especially those bearing the stigmata of degenera-

tion, there may be evidence of sexual perversion. The "damnable iteration" with which writers in our ranks "dish up" this unpleasant subject is proof positive that not all prophets speak to edification.

In females it is common to find a tender ovary, and painful or irregular menstruation.

In all forms of neurasthenia the condition of the urine is important. Many cases are complicated with the symptoms of the condition known as lithæmia, and so marked may this be that some have indeed made a special form of lithæmic neurasthenia. Polyuria may be present, but is more common in hysteria. With disturbed digestion the urates and oxalates may be in excess.

Diagnosis.—While in the majority of cases the diagnosis can readily be made, still there are instances in which it is very difficult. Neurasthenia overlaps hypochondria and hysteria on the one hand, and the psychoses and degenerative diseases of the nervous system on the other. The term has in the past been altogether too loosely used. Simple local disturbances and temporary general disturbances the result of sudden overexertion should scarcely be diagnosed as neurasthenia. Only when we have before us a clinical picture indicating general weakness of the nervous system in addition to the local disturbances, no matter how pronounced they are, is the diagnosis justifiable. Charcot has designated as neurasthenic stigmata certain fundamental and typical symptoms, such as the pain and pressure in the head, the disturbances of sleep, the rhachialgia and spinal hyperæsthesia, the muscular weakness, the nervous dyspepsia, the disturbances of the genital organs, and the typical mental phenomena (irritable humor, psychic depression, feelings of anxiety, intellectual fatigue, incapacity of decision, and the like). In addition to these cardinal symptoms of the disease, he described as secondary or accessory symptoms the feelings of dizziness and vertigo, the neurasthenic asthenopia, the circulatory, respiratory, secretory, and nutritive disturbances, disturbances of motility and sensation, the fever of neurasthenia, and neurasthenic idiosyncrasies. The anxiety conditions and various phobias, as well as the different varieties of tic and the occupation neuroses when they accompany neurasthenia, are regarded as complications dependent in the majority of instances upon faulty heredity. I must agree with Binswanger in emphasizing the importance for the diagnosis of the peculiar intellectual and emotional condition of the patient, as well as the disturbances of sleep.

Neurasthenia is a disease above all others which has to be diagnosed from the subjective statements of the patient, and from an observation of his general behavior rather than from the physical examination. The physical examination is of the highest importance in excluding other diseases likely to be confounded with it. That somatic changes occur and that physical signs are often to be made out is very true, and we owe to Löwenfeld especially a careful discussion of these points, but there is nothing typical or pathognomonic in these objective changes.

The hypochondriac differs from the neurasthenic in the excessive psychic distortion of the pathological sensations to which he is subject. He is the victim of actual delusions regarding his condition.

The confusion of neurasthenia with hysteria is still more frequent; in women especially a diagnosis of hysteria is often made when in reality the condition is one of neurasthenia. In the absence of hysterical paroxysms, of crises, and of those marked emotional and intellectual characteristics of the hysterical individual the diagnosis of hysteria should not be made. Of course, in many of the cases of hysteria definite hysterical stigmata (hysterical paralyses, convulsions, contractures, anæsthesias, alterations in the visual field, etc.) are present, and the diagnosis is not difficult.

Epilepsy is not likely to be confounded with neurasthenia if there be definite epileptic attacks, but the cases of *petit mal* may be puzzling.

The onset of exophthalmic goitre may be mistaken for neurasthenia, especially if there be no exophthalmos at the beginning. The emotional disturbances and the irritability of the heart may mislead the physician. In pronounced cases of nervous prostration the differential diagnosis from the various psychoses may be extremely difficult.

The two forms of organic disease of the nervous system with which neurasthenia is most likely to be confounded are tabes and general paresis. The symptoms of the spinal form of neurasthenia may resemble those of the former disease, while the symptoms of the psychic or cerebral form of neurasthenia may be very similar to those of general paresis. The diagnosis, as a rule, presents no difficulty if the physician be careful to make a thorough routine examination. It is only the superficial study of a case that is likely to lead one astray. In tabes especially a consideration of the sensory disturbances, of the deep reflexes, and of the pupillary findings will always establish the presence or absence of the disease. In general paresis there is sometimes more difficulty. The onset of general paresis is often characterized by the appearance of symptoms quite like those of ordinary neurasthenia, and the family physician may entirely overlook the grave nature of the malady. The mistake in the other direction is, however, perhaps just as common. A physician who once or twice has seen a case of general paresis develop out of what appeared to be one of pronounced neurasthenia is too prone afterward to suspect every neurasthenic to be developing the malign affection. The most marked symptoms, however, of psychic exhaustion do not justify a diagnosis of general paresis even when the history is suspicious, unless along with it definite paresis of the facial or muscles of articulation or of the pupils exist. A history of syphilis or of chronic alcoholism or morphinism associated with severe psychic exhaustion should, of course, put one always on his guard, and the physician should be sharply on the lookout for the appearance of intellectual defects, paraphasia, facial paresis, and sluggishness of the pupils.

Treatment.—*Prophylaxis.*—Many patients come under our care a generation too late for satisfactory treatment, and it may be impossible to restore the exhausted capital. The greatest care should be taken in the rearing of children of neuropathic predisposition. From a very early age they should be submitted to a process of "psychic hardening," every effort being made to strengthen the bodily and mental condition. Even in infancy the child should not be pampered. Later on the greatest care should

be exercised with regard to food, sleep, and school work. Complaints of children should not be too seriously considered.

Much depends upon the example set by the parents. A restless, emotional, constantly complaining mother will rack the nervous system of a delicate child. In some instances, for the welfare of a developing boy or girl, the physician may find it necessary to advise its removal from home.

Neurotic children are especially liable during development to fits of temper and of emotional disturbance. These should not be too lightly considered. Above all, violent chastisement in such cases is to be avoided, and loss of temper on the part of the parent or teacher is particularly pernicious for the nervous system of the child. Where possible, in such instances, the best treatment is to put the obstreperous child immediately to bed, and if the excitement and temper continue a warm bath followed by a cool douch may be effective. If he be put to bed after the bath sleep soon follows.

Special attention is necessary at puberty in both boys and girls. If there be at this period any marked tendency to emotional disturbance or to intellectual weakness the child should be removed from school and every care taken to avoid unfavorable influences.

Personal Hygiene.—Throughout life individuals of neuropathic predisposition should obey scrupulously certain hygienic and prophylactic rules. Intellectual work especially should be judiciously limited and should alternate frequently with periods of repose. Excitement of all kinds should of course be avoided, and such individuals will do well to be abstemious in the use of tobacco, tea, coffee, and alcohol, if, indeed, they be permitted to use these substances at all. The habit, happily in this country becoming very common, of taking at least once a year a prolonged holiday away from the ordinary environment, in the woods, in the mountains, or at the seashore, should be urgently enjoined upon every neuropathic individual. In many instances it is found to be the greatest relief and rest if the patient can take his holiday away from his relatives.

During ordinary life nervous people should, during some portion of each day, pay rational attention to the body. Cold baths, swimming, exercises in the gymnasium, gardening, golf, lawn tennis, cricket, hunting, shooting, rowing, sailing, and bicycling are of value in maintaining the general nutrition. Such exercises are, of course, to be recommended only to individuals physically equal to them. If neurasthenia be once well developed the greatest care must be observed in the ordering of exercise. Many nervous girls have been completely broken down by following injudicious advice with regard to long walks.

Treatment of the Condition.—The treatment of neurasthenia when once established presents a varied problem to the thoughtful physician. Every case must be handled upon its own merits, no two, as a rule, requiring exactly the same methods. In general it will be the aim of the medical adviser to remove the patient as far as possible from the influences which have led to his downfall, and to restore to normal the nervous mechanisms which have been weakened by injurious influences. The general character

of the individual, his physical and social status must of course be considered, and the therapeutic measures carefully adjusted to these.

Above all, the physician must first gain the confidence of his patient, and this he will not do if he be inattentive to the complaints of the individual, especially at first, or if he rudely tell the patient before he has carefully examined him and observed him for some time that his troubles are imaginary. As has been said, it is education more than medicine that these patients need, but the patients themselves do not wish to be educated; they come to the physician to be treated, and the educating process has to be disguised.

The diagnosis having been settled, the physician may assure the patient that with prolonged treatment, during which his coöperation with the physician is absolutely essential, he may expect to get well. He must be told that much depends upon himself and that he must make a vigorous effort to overcome certain of his tendencies, and that all his strength of will will be needed to further the progress of the cure. In the case of business or professional men, in whom the condition develops as a result of overwork or overstudy, it may be sufficient to enjoin absolute rest with change of scene and diet. A trip abroad, with a residence for a month or two in Switzerland, or, if there are symptoms of nervous dyspepsia, a residence at one of the Spas will usually prove sufficient. The excitement of the large cities abroad should be avoided. The longer the disease has lasted and the more intense the symptoms have been, the longer the time necessary for the restoration of health. In cases of any severity the patient must be told that at least six months' complete absence from business, under strict medical guidance, will be necessary. Shorter periods may of course be of benefit, which, however, as a rule, will be only temporary.

It will be wise in very many cases to treat the individual for a few weeks at least in a hospital or other institution before sending him away on a journey. In this preliminary treatment the greatest tact is required on the part of the medical attendant and nurse. The patient should not see the doctor too often after the first careful examination, although he should of course receive regular visits from him. The physician will make a mistake if he responds to frequent calls on the part of the patient between the periods of his regular visits. The choice of a nurse is by no means an easy matter. That she should be healthy, strong, and by no means nervous herself are among the first considerations. Sallow-faced, emotional, emaciated women can only do harm if detailed to the care of a nervous patient.

It will often be found advisable to make out a daily programme, which shall occupy almost the whole time of the patient. At first he need know nothing about this, the case being given over entirely to the nurse. As improvement advances, moderate physical and intellectual exercises, alternating frequently with rest and the administration of food, may be undertaken. Some one hour of the day may be left free for reading, correspondence, conversation, and games. In some instances the writing of letters is particularly harmful to the patient and must be prohibited or limited. Cultured individuals may find benefit from attention to drawing, painting, mod-

elling, translating from a foreign language, the making of abstracts, etc., for short periods in the day.

In not a few cases, including a large proportion of neurasthenic women, a systematic Weir Mitchell treatment rigidly carried out should be tried (see Hysteria). For obstinate and protracted cases, particularly if combined with the chloral or morphia habit, no other plan is so satisfactory. The patient must be isolated from his friends, and any regulations undertaken must be strictly adhered to, the consent of the patient and his family having first been gained. If the case responds well to the treatment there should be a gain of from 2 to 4 pounds per week. The benefit is often extraordinary, individuals increasing in weight as much as from 50 to 80 pounds in the course of twelve weeks. The treatment of the gastric and intestinal symptoms so important in this condition has already been considered. For the irregular pains, particularly in the back and neck, the thermo-cautery is invaluable.

Hydrotherapy is indicated in nearly every case if it can be properly applied. Much can be done at home or in an ordinary hospital, but for systematic hydrotherapeutic treatment residence in a suitable sanitarium is necessary. I have found the wet pack of especial value. Particularly at night in cases of sleeplessness it is perhaps the best remedy against insomnia we have. Some patients gain rapidly in weight through the systematic use of the wet pack. Salt baths are more helpful to some patients. The various forms of douches, partial packs, foot baths, etc., may be valuable in individual cases. The Scotch douche is often invigorating in the milder cases.

Electrotherapy is of some value, though only in combination with psychic treatment and hydrotherapy. General and local faradization, galvanic electricity, and Franklinization may be used; in every case, however, with great caution and only by skilled operators.

Treatment by drugs should be avoided as much as possible. They are of benefit chiefly in the combating of single symptoms. A placebo is sometimes necessary for its psychic effect. Alcohol, morphia, chloral, or cocaine should never be given. The family physician is often responsible for the development of a drug habit. I have been repeatedly shocked by the loose, careless way in which physicians inject morphia for a simple headache or a mild neuralgia.

General tonics may be helpful, especially if the individual be anæmic. Arsenic and more often iron are then indicated. The value of phosphorus has been exaggerated. For the severer pains and nervous attacks some sedative may occasionally be necessary, especially at the beginning of the treatment. The bromides, especially a mixture of the salts of ammonium, potassium, and sodium may here be given with advantage. An occasional dose of phenacetin, antipyrin, or salipyrin may be required, but the less of these substances we can get along with the better. For the relief of sleeplessness all possible measures should be resorted to before the employment of drugs. The wet pack will usually suffice. If absolutely necessary to give a drug, sulphonal, trional, or amylene hydrate may be employed.

In cases in which the anxiety conditions are disturbing, the cautious use

of opium in pill form may be necessary, since, as in the psychoses, opium here will sometimes yield permanent relief. A prolonged treatment with opium is, however, never necessary in neurasthenia.

XIII. THE TRAUMATIC NEUROSES

(*Railway Brain and Railway Spine; Traumatic Hysteria*).

Definition.—A morbid condition following shock which presents the symptoms of neurasthenia or hysteria or of both. The condition is known as "railway brain" and "railway spine."

Erichsen regarded the condition as the result of inflammation of the meninges and cord, and gave it the name railway spine. Walton and J. J. Putnam, of Boston, were the first to recognize the hysterical nature of many of the cases, and to Westphal's pupils we owe the name traumatic neurosis. For an excellent discussion of the whole question the reader is referred to Pearce Baily's recent work, On Accident and Injury; their Relation to Diseases of the Nervous System.

Etiology.—The condition follows an accident, often in a railway train, in which injury has been sustained, or succeeds a shock or concussion, from which the patient may apparently not have suffered in his body. A man may appear perfectly well for several days, or even a week or more, and then develop the symptoms of the neurosis. Bodily shock or concussion is not necessary. The affection may follow a profound mental impression; thus, an engine-driver ran over a child, and received thereby a very severe shock, subsequent to which the most pronounced symptoms of neurasthenia developed. Severe mental strain combined with bodily exposure may cause it, as in a case of a naval officer who was wrecked in a violent storm and exposed for more than a day in the rigging before he was rescued. A slight blow, a fall from a carriage or on the stairs may suffice.

Symptoms.—The cases may be divided into three groups: simple neurasthenia, cases with marked hysterical manifestations, and cases with severe symptoms indicating or simulating organic disease.

(*a*) *Simple Traumatic Neurasthenia.*—The first symptoms usually develop a few weeks after the accident, which may or may not have been associated with an actual trauma. The patient complains of headache and tired feelings. He is sleepless and finds himself unable to concentrate his attention properly upon his work. A condition of nervous irritability develops, which may have a host of trivial manifestations, and the entire mental attitude of the person may for a time be changed. He dwells constantly upon his condition, gets very despondent and low-spirited, and in extreme cases melancholia may develop. He may complain of numbness and tingling in the extremities, and in some cases of much pain in the back. The bodily functions may be well performed, though such patients usually have, for a time at least, disturbed digestion and loss in weight. The physical examination may be entirely negative. The reflexes are slightly increased, as in ordinary neurasthenia. The pupils may be un-

equal; the cardio-vascular changes already described in neurasthenia may be present in a marked degree. According as the symptoms are more spinal or more cerebral, the condition is known as railway brain or railway spine.

(2) *Cases with Marked Hysterical Features.*—Following an injury of any sort, neurasthenic symptoms, like those described above, may develop, and in addition symptoms regarded as characteristic of hysteria. The emotional element is prominent, and there is but slight control over the feelings. The patients have headache, backache, and vertigo. A violent tremor may be present, and indeed constitutes the most striking feature of the case. I have recently seen an engineer who developed subsequent to an accident a series of nervous phenomena, but the most marked feature was an excessive tremor of the entire body, which was specially manifest during emotional excitement. The most pronounced hysterical symptoms are the sensory disturbances. As first noted by Putnam and Walton, hemianæsthesia may occur as a sequence of traumatism. This is a common symptom in France, but rare in England and in this country. Achromatopsia may exist on the anæsthetic side. A second, more common, manifestation is limitation of the field of vision, similar to that which occurs in hysteria.

Remarkable disturbances may develop in some of these cases. A few months ago I saw a man who had been struck by an electric car, whose chief symptom was an extraordinary increase in the number of respirations. He was a stout, powerfully built man, and presented practically no other symptom than dyspnœa of the most extreme grade. At the time of observation his respirations were over 130 per minute, and he stated that they had been counted at over 150.

(3) *Cases in which the Symptoms suggest Organic Disease of the Brain and Cord.*—As a result of spinal concussion, without fracture or external injury, there may subsequently develop symptoms suggestive of organic disease, which may come on rapidly or at a late date. In a case reported by Leyden the symptoms following the concussion were at first slight and the patient was regarded as a simulator, but finally the condition became aggravated and death resulted. The post mortem showed a chronic pachymeningitis, which had doubtless resulted from the accident. The cases in this group about which there is so much discussion are those which display marked sensory and motor changes. Following an accident in which the patient has not received external injury a condition of excitement may develop within a week or ten days; he complains of headache and backache, and on examination sensory disturbances are found, either hemianæsthesia or areas on the skin in which the sensation is much benumbed; or painful and tactile impressions may be distinctly felt in certain regions, and the temperature sense is absent. The distribution may be bilateral and symmetrical in limited regions or hemiplegic in type. Limitation of the field of vision is usually marked in these cases, and there may be disturbance of the senses of taste and smell. The superficial reflexes may be diminished; usually the deep reflexes are exaggerated. The pupils may be unequal; the motor disturbances are variable. The French writers describe cases of

monoplegia with or without contracture, symptoms upon which Charcot lays great stress as a manifestation of profound hysteria. The combination of sensory disturbances—anæsthesia or hyperæsthesia—with paralysis, particularly if monoplegic, and the occurrence of contractures without atrophy and with normal electrical reactions, may be regarded as distinctive of hysteria.

In rare cases following trauma and succeeding to symptoms which may have been regarded as neurasthenic or hysterical, there are organic changes which may prove fatal. That this sequence occurs is demonstrated clearly by recent post-mortem examinations. The features upon which the greatest reliance can be placed as indicating organic change are optic atrophy, bladder symptoms, particularly in combination with tremor, paresis, and exaggerated reflexes.

The anatomical changes in this condition have not been very definite. When death follows spinal concussion within a few days there may be no apparent lesion, but in some instances the brain or cord has shown punctiform hæmorrhages. Edes has reported 4 cases in which a gradual degeneration in the pyramidal tracts followed concussion or injury of the spine; but in all these cases there was marked tremor and the spinal symptoms developed early or followed immediately upon the accident. Post mortems upon cases in which organic lesions have supervened upon a traumatic neurosis are extremely rare. Bernhardt reports an instance of a man, aged thirty-three, who in 1886 received a kick from a horse on the epigastrium and subsequently developed the symptom-complex of neurasthenia and hysteria with attacks of vertigo and great psychical depression. He afterward had more marked mental symptoms and attacks of unconsciousness. He committed suicide and the brain and cord showed a beginning multiple sclerosis in the white matter, which was possibly associated with an advanced grade of arterio-sclerosis. In a second case a man, aged forty-two, received a shock in a railway accident in July, 1884. He was rendered unconscious and had a slight injury in the buttock region. In a few weeks symptoms of traumatic neurosis developed, particularly great depression of spirits, with headache and sensory disturbances in the feet and hands. Tremor and great weakness were complained of when he attempted to work. There was no increase in the reflexes. The case was regarded as an instance of simulation and a defect in objective symptoms favored this view. Subsequently this judgment was reversed, but he did not improve. He died in January, 1889, with symptoms of cardiac dyspnœa. Macroscopically the brain and cord appeared normal. There was extreme arterio-sclerosis, particularly of the vessels of the brain and cord. In the latter there were scattered areas of degeneration in the white substance, and degeneration in the sympathetic ganglia.

I have entered somewhat fully into this question because of its extreme importance and on account of the paucity of the observations upon cases which have subsequently developed symptoms of organic disease. Examples of it are extremely rare. So far as I know no case with autopsy has been reported in this country, nor have I seen an instance in which the clinical features pointed to an organic disease which had followed upon a traumatic neurosis.

Diagnosis.—A condition of fright and excitement following an accident may persist for days or even weeks, and then gradually pass away. The symptoms of neurasthenia or of hysteria which subsequently develop present nothing peculiar and are identical with those which occur under other circumstances. Care must be taken to recognize simulation, and, as in these cases the condition is largely subjective, this is sometimes extremely difficult. In a careful examination a simulator will often reveal himself by exaggeration of certain symptoms, particularly sensitiveness of the spine, and by increasing voluntarily the reflexes. Maunkopff suggests as a good test to take the pulse-rate before, during, and after pressure upon an area said to be painful. If the rate is quickened, it is held to be proof that the pain is real. This is not, however, always the case. It may require a careful study of the case to determine whether the individual is honestly suffering from the symptoms of which he complains. A still more important question in these cases is, Has the patient organic disease? The symptoms given under the first two groups of cases may exist in a marked degree and may persist for several years without the slightest evidence of organic change. Hemianæsthesia, limitation of the field of vision, monoplegia with contracture, may all be present as hysterical manifestations, from which recovery may be complete. In our present knowledge the diagnosis of an organic lesion should be limited to those cases in which optic atrophy, bladder troubles, and signs of sclerosis of the cord are well marked—indications either of degeneration of the lateral columns or of multiple sclerosis.

Prognosis.—A majority of patients with traumatic hysteria recover. In railway cases, so long as litigation is pending and the patient is in the hands of lawyers the symptoms usually persist. Settlement is often the starting-point of a speedy and perfect recovery. I have known return to health after the persistence of the most aggravated symptoms with complete disability of from three to five years' duration. On the other hand, there are a few cases in which the symptoms persist even after the litigation has been closed; the patient goes from bad to worse and psychoses develop, such as melancholia, dementia, or occasionally progressive paresis. And, lastly, in extremely rare cases, organic lesions may develop as a sequence of the traumatic neurosis.

The function of the physician acting as medical expert in these cases consists in determining (*a*) the existence of actual disease, and (*b*) its character, whether simple neurasthenia, severe hysteria, or an organic lesion. The outlook for ultimate recovery is good except in cases which present the more serious symptoms above mentioned. Nevertheless, it must be borne in mind that traumatic hysteria is one of the most intractable affections which we are called upon to treat. In the treatment of the traumatic neuroses the practitioner may be guided by the principles laid down in the preceding chapter, in which the treatment of neurasthenia in general has been described.

XIV. OTHER FORMS OF FUNCTIONAL PARALYSIS.

I. Periodical Paralysis.

I have already referred to the remarkable periodical paralysis of the ocular muscles, which may recur at intervals for many years. There is a form of periodical paralysis involving the general muscles, which may recur with great regularity, and which is also a "family" affection. Goldflam has described a family in which twelve members were affected with this disease, the heredity being through the mother. In this country E. W. Taylor has described in one family 11 cases in five generations.

The clinical picture is very much alike in all the recorded cases. The paralysis involves, as a rule, the arms and legs. It comes on when the patients are in full health, and without any apparent cause, often during sleep. Sometimes it begins with weakness in the limbs, a sensation of weariness and sleepiness, not often with sensory symptoms. The paralysis is usually complete within the first twenty-four hours, beginning in the legs, to which in rare instances it is confined. The muscles of the neck are sometimes involved, and occasionally those of the tongue and pharynx. The cerebral nerves and the special senses are, as a rule, uninvolved. The attacks are afebrile, sometimes with low temperatures and slow pulse. The deep reflexes are reduced, sometimes abolished, and the skin reflexes may be feeble. One of the most remarkable features is the extraordinary reduction or complete abolition of the faradic excitability, both of muscles and of nerves.

Improvement begins sometimes in the course of a few hours or after a day or two, and the paralysis disappears completely, and the patient is perfectly well. As mentioned, the attacks may recur every few weeks, in some instances even daily; more commonly, an interval of one or two weeks elapses between the attacks. There may be signs of acute dilatation of the heart during the attack. After the fiftieth year the attacks usually cease.

II. Astasia; Abasia.

These terms, indicating respectively inability to stand and inability to walk, have been applied by Charcot and Blocq to diseased conditions characterized by loss of the power of standing or of walking, with retention of muscular power, coördination, and sensation. Blocq's definition is as follows: "A morbid state in which the impossibility of standing erect and walking normally is in contrast with the integrity of sensation, of muscular strength, and of the coördination of the other movements of the lower extremities." The condition forms a symptom group, not a morbid entity, and is probably a functional neurosis. Knapp in his monograph analyzes the 50 cases reported in the literature. Twenty-five of these were in men, 25 in women. In 21 cases hysteria was present; in 3, chorea; in 2, epilepsy; and in 4, intention psychoses. As a rule, the patients, though able to move the feet and legs perfectly when in bed, are either unable to walk properly or cannot stand at all. The disturbances have been very varied,

and different forms have been recognized. The commonest, according to Knapp's analysis of the recorded cases, is the paralytic, in which the legs give out as the patient attempts to walk and "bend under him as if made of cotton." "There is no rigidity, no spasm, no incoördination. In bed, sitting, or even while suspended, the muscular strength is found to be good." Other cases are associated with spasm or ataxia; thus there may be movements which stiffen the legs and give to the gait a somewhat spastic character. In other instances there are sudden flexions of the legs, or even of the arms, or a saltatory, spring-like spasm. In a majority of the cases it is a manifestation of a neurosis allied to hysteria.

The cases, as a rule, recover, particularly in young persons. Relapses are not uncommon. The rest treatment and static electricity should be employed.

VIII. VASO-MOTOR AND TROPHIC DISORDERS.

I. RAYNAUD'S DISEASE.

Definition.—A vascular disorder, probably dependent upon vaso-motor influences, characterized by three grades of intensity: (*a*) Local syncope, (*b*) local asphyxia, and (*c*) local or symmetrical gangrene.

Local Syncope.—This condition is seen most frequently in the extremities, producing the condition known as dead fingers or dead toes. It is analogous to that produced by great cold. The entire hand may be affected with the fingers; more commonly only one or more of the fingers. This feature of the disease rarely occurs alone, but is generally associated with local asphyxia. The common sequence is as follows: On exposure to slight cold or in consequence of some emotional disturbance the fingers become white and cold, or both fingers and toes are affected. The pallor may continue for an indefinite time, though usually not more than an hour or so; then gradually a reaction follows and the fingers get burning hot and red. This does not necessarily occur in all the fingers together; one finger may be as white as marble, while the adjacent ones are of a deep red or plum color.

Local Asphyxia.—Chilblains form the mildest grade of this condition. It usually follows the local syncope, but it may come on independently. The fingers and toes are oftenest affected, next in order the ears; more rarely portions of the skin on the arms and legs. During an attack the fingers alone, sometimes the hands, also swell and become intensely congested. In the most extreme grade the fingers are perfectly livid, and the capillary circulation is almost stagnant. The swelling causes stiffness and usually pain, not acute, but due to the tension and distention of the skin. Sometimes there is marked anæsthesia. Pain of a most excruciating kind may be present. Attacks of this sort may recur for years, and be brought on by the slightest exposure to cold or in consequence of disturbances, either mental or, in some instances, gastric. Apart from this unpleasant symp-

tom the general health may be very good. The condition is always worse during the winter, and may be present only when the external temperature is low.

Local or Symmetrical Gangrene.—The mildest grade of this condition follows the local asphyxia, in the chronic cases of which small necrotic areas are sometimes seen at the tips of the fingers. Sometimes the pads of the fingers and of the toes are quite cicatricial from repeated slight losses of this kind. So also when the ears are affected there may be superficial loss of substance at the edge. The severer cases, which terminate in extensive gangrene, are fortunately rare.

In an attack the local asphyxia persists in the fingers. The terminal phalanges, or perhaps the end of only one finger, become black, cold, and insensible. The skin begins to necrose and superficial gangrenous blebs appear. Gradually a line of demarkation shows itself and a portion of one or more of the fingers sloughs away. The resulting loss of substance is much less than the appearance of the hand or foot would indicate, and a condition which looks as if the patient would lose all the fingers or half of a foot may result perhaps in only a slight superficial loss in the phalanges. In severer cases the greater portion of a finger or the tip of the nose may be lost. Occasionally the disease is not confined to the extremities, but affects symmetrical patches on the limbs or trunk, and may pass on to rapid gangrene. These severe types of cases occur particularly in young children, and death may result within three or four days. The attacks are usually very painful, and the motion of the part is much impaired. In some cases numbness and tingling persist for a long time.

The climax of this series of neuro-vascular changes is seen in the remarkable instances of extensive multiple gangrene. They are most common in children, and may progress with frightful rapidity. In the Medico-Chirurgical Society's Transactions, vol. xxii, there is an extraordinary case reported, in which the child, aged three, lost in this way both arms above the elbow, and the left leg below the knee. There also had been a spot of local gangrene on the nose. Spontaneous amputation occurred, and the child made a complete recovery. The cases are more frequent than has been supposed, and an illustration is given by Weeks, of Marion, Ohio, in which the boy had rheumatic pains in the legs, and purpuric blotches developed before the gangrene began (Medico-Surgical Bulletin, July 1, 1894).

There are remarkable concomitant symptoms in Raynaud's disease to which a good deal of attention has been paid of late years. Hæmoglobinuria may develop during an attack, or may take the place of an outbreak. In such instances the affection is usually brought on by cold weather. In a case reported by H. M. Thomas from my clinic, Raynaud's disease occurred for three successive winters and always in association with hæmoglobinuria. The attacks were sometimes preceded by a chill. Several cases of the kind are found in Barlow's appendix to his translation of Raynaud's paper for the New Sydenham Society. The onset with a chill, as in the case just mentioned, has doubtless given rise to the idea that the disease is in some way associated with ague. Cerebral symptoms, particularly mental torpor

and transient loss of consciousness, have also been noticed in some cases. The case just mentioned with hæmoglobinuria had epilepsy with the attacks. Exposure on a cold day would bring on an epileptic seizure with the local asphyxia and bloody urine. Another patient, the subject for years of Rayna[illegible] iplegia on one side or t[illegible] ce the second edition [illegible] sionally joint affection[illegible] the phalangeal arti[illegible] developed, and Ba[illegible] Peripheral neuritis[illegible]

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Defin[illegible] usually one or more extremities [illegible], made far worse if the parts hang down" (Weir Mitchell). The name signifies a painful, red extremity.

Symptoms.—In 1872 (Phila. Med. Times, November 23d), in a lecture on certain painful affections of the feet, Weir Mitchell described the case of a sailor, aged forty, who after an African fever began to have "dull, heavy pains, at first in the left and soon after in the right foot. There was no swelling at first. When at rest he was comfortable and the feet were not painful. After walking the feet were swollen. They scarcely pitted on pressure, but were purple with congestion; the veins were everywhere singularly enlarged, and the arteries were throbbing visibly. The whole

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JANUARY 6, 1900]

MEDICAL

Erythromelalgy.—ROSIN.* In order to diagnose erythromelalgy there must be present pain, which should be intense and constant, and a redness and swelling of the extremities, sweating, a feeling of heat in the members affected, a definite increase in temperature of the parts, and a definite hyperalgesia. The affection is seldom confined to one extremity. Characteristic is the chronic course of the disease. There may be variations to this clinical picture. The case described by the author is as follows: Patient, 34 years old; no nervous heredity. The patient [illegible] intellectually backward, and has gradually developed into a condition of idiocy. For three and one-half years the mother noticed a swelling in both feet, more noticeable in the evening than in the morning. There is great pain in the extremities. It was further noticed that the painful parts were deeply red in color, and were covered with sweat. The feet were warmer to the touch than normal. On examination, these symptoms were found objectively present: The temperature of the swollen and reddened portions was found 3° higher than the normal, uncolored portions. Hyperalgesia was found. The muscles otherwise were normal. There is no doubt that the case is one of erythromelalgy in an idiot.

where there has been stercoraceous vomiting, the stomach should be washed out with a tube and funnel with warm brandy and water until quite clean. The patient should then be wrapped from head to foot in warm flannel or wool and lie between two hot bottles, with another between his legs. With feeble individuals, if not in all, it is desirable that hypodermic transfusion of warm normal saline solution should be provided for throughout the operation to make up for the fluid lost by vomiting. In the matter of operation, everything which saves time is of the utmost importance. The loop must be packed round with aseptic gauze most carefully and it must be emptied by pressure and clamped. In regard to the after-treatment of hernia, hypodermic transfusion of saline solution twice a day to the extent of a pint or so each time is an important factor. Nutritive enemata every four hours and hot water enemata every four hours, alternating with the last, have for many years been a routine treatment in all the writer's cases of abdominal operations.

Some Practical Notes on Diseases of the Rectum.—L. H. ADLER, JR.,* refers briefly to some practical points in the consideration of some of the forms of rectal diseases. Congenital malformations of the [illegible] rectum occur often enough [illegible]

and transient loss of consciousness, have also been noticed in some cases. The case just mentioned with hæmoglobinuria had epilepsy with the attacks. Exposure on a cold day would bring on an epileptic seizure with the local asphyxia and bloody urine. Another patient, the subject for years of Raynaud's disease, has had many attacks of transient hemiplegia on one side or the other, when on the right side with aphasia. Since the second edition of this work was issued she died in an attack. Occasionally joint affections develop, particularly anchylosis and thickening of the phalangeal articulations. Southey has reported a case in which mania developed, and Barlow an instance in which the woman had delusions. Peripheral neuritis has been found in several cases.

The *pathology* of this remarkable disease is still obscure. Raynaud suggested that the local syncope was produced by contraction of the vessels, which seems likely. The asphyxia is dependent upon dilatation of the capillaries and small veins, probably with the persistence of some degree of spasm of the smaller arteries. There are two totally different forms of congestion, which may be shown in adjacent fingers; one may be swollen, of a vivid red color, extremely hot, the capillaries and all the vessels fully distended, and the anæmia produced by pressure may be instantaneously obliterated; the adjacent finger may be equally swollen, absolutely cyanotic, stone cold, and the anæmia produced by pressure takes a long time to disappear. In the latter case the arterioles are probably still in a condition of spasm.

Treatment.—In many cases the attacks recur for years uninfluenced by treatment. Mild attacks require no treatment. In the severer forms of local asphyxia, if in the feet, the patient should be kept in bed with the legs elevated. The toes should be wrapped in cotton-wool. The pain is often very intense and may require morphia. Carefully applied, systematic massage of the extremities is sometimes of benefit. Galvanism may be tried. Barlow advises immersing the affected limb in salt water and placing one electrode over the spine and the other in the water. Nitroglycerin has been warmly recommended by Cates.

II. ERYTHROMELALGIA (*Red Neuralgia*).

Definition.—"A chronic disease in which a part or parts—usually one or more extremities—suffer with pain, flushing, and local fever, made far worse if the parts hang down" (Weir Mitchell). The name signifies a painful, red extremity.

Symptoms.—In 1872 (Phila. Med. Times, November 23d), in a lecture on certain painful affections of the feet, Weir Mitchell described the case of a sailor, aged forty, who after an African fever began to have "dull, heavy pains, at first in the left and soon after in the right foot. There was no swelling at first. When at rest he was comfortable and the feet were not painful. After walking the feet were swollen. They scarcely pitted on pressure, but were purple with congestion; the veins were everywhere singularly enlarged, and the arteries were throbbing visibly. The whole

foot was said to be aching and burning, but above the ankle there was neither swelling, pain, nor, flushing." As the weather grew cool he got relief. Nothing seemed to benefit him. This brief summary of Mitchell's first case gives an accurate clinical picture of the disease. His second communication, On a Rare Vaso-motor Neurosis of the Extremities, appeared in the Am. Jour. of the Medical Sciences for July, 1878, while in his Clinical Lessons on Nervous Diseases, 1897, will be found additional observations.

The disease is rare. Rost states that there are only about 40 instances in the literature. The feet are much more often affected than the hands. The pain may be of the most atrocious character. It is usually, but not always, relieved by cool weather; in one of my cases the winter aggravates the trouble. In a few cases (Elsner, Dehio, Rolleston) the affection has been complicated with Raynaud's disease.

Mitchell speaks of it as a "painful nerve-end neuritis." Dehio suggests that there may be irritation in the cells of the ventral horns of the cord at certain levels. Excision of the nerves passing to the parts has been followed by relief. In one of Mitchell's cases gangrene of the foot followed excision of four inches of the musculo-cutaneous nerve and stretching of the posterior tibial. Sclerosis of the arteries was found.

III. ANGIO-NEUROTIC ŒDEMA.

Definition.—An affection characterized by the occurrence of local œdematous swellings, more or less limited in extent, and of transient duration. Severe colic is sometimes associated with the outbreak. There is a marked hereditary disposition in the disease.

Symptoms.—The œdema appears suddenly and is usually circumscribed. It may appear in the face; the eyelid is a common situation; or it may involve the lips or cheek. The backs of the hands, the legs, or the throat may be attacked. Usually the condition is transient, associated perhaps with slight gastro-intestinal distress, and the affection is of little moment. There may be a remarkable periodicity in the outbreak of the œdema. In Matas' case this periodicity was very striking; the attack came on every day at eleven or twelve o'clock. The disease may be hereditary through many generations. In the family whose history I reported, five generations had been affected, including twenty-two members. The swellings appear in various parts; only rarely are they constant in one locality. The hands, face, and genitalia are the parts most frequently affected. Itching, heat, redness, or in some instances, urticaria may precede the outbreak. Sudden œdema of the larynx may prove fatal. Two members of the family just referred to died of this complication. In one member of this family, whom I saw repeatedly in attacks, the swellings came on in different parts; for example, the under lip would be swollen to such a degree that the mouth could not be opened. The hands enlarge suddenly, so that the fingers cannot be bent. The attacks recur every three or four weeks. Accompanying them are usually gastro-intestinal attacks, severe colic, pain,

nausea, and sometimes vomiting. It is quite possible that some of the cases of Leyden's intermittent vomiting may belong to this group. The colic is of great intensity and usually requires morphia. Arthritis apparently does not occur. Periodic attacks of cardialgia have also been met with during the outbreak of the œdema. Hæmoglobinuria has occurred in several cases.

The disease has affinities with urticaria, the giant form of which is probably the same disease. There is a form of severe purpura, often with urticarial manifestations, which is also associated with marked gastro-intestinal crises, and it is interesting to note that Schlesinger has reported a case in which a combination of erythromelalgia, Raynaud's disease, and acute œdema occurred. Quincke regards the condition as a vaso-motor neurosis, under the influence of which the permeability of the vessels is suddenly increased. Milroy, of Omaha, has described cases of hereditary œdema, twenty-two individuals in six generations, in which there existed from birth a solid œdema of one or of both legs, without any special inconvenience or any progressive increase of the disease.

Some years ago I described a remarkable vaso-motor neurosis characterized by *swelling and tumefaction of the whole arm on exertion.* My patient was a man, healthy in every other respect. Recently in Philadelphia a similar case has been observed. On the supposition that there might be pressure on the axillary vessels these were exposed, but nothing was found.

The *treatment* is very unsatisfactory. In the cases associated with anæmia and general nervousness, tonics, particularly large doses of strychnia, do good; but too often the disease resists all treatment. I have seen great improvement follow the prolonged use of nitroglycerin.

IV. FACIAL HEMIATROPHY.

An affection characterized by progressive wasting of the bones and soft tissues of one side of the face. The atrophy starts in childhood, but in a few cases has not come on until adult life. Perhaps after a trifling injury or disease the process begins, either diffusely or more commonly at one spot on the skin. It gradually spreads, involving the fat, then the bones, more particularly the upper jaw, and last and least the muscles. The wasting is sharply limited at the middle line, and the appearance of the patient is very remarkable, the face looking as if made up of two halves from different persons. There is usually change in the color of the skin and the hair falls. Owing to the wasting of the alveolar processes the teeth become loose and ultimately drop out. The eye on the affected side is sunken, owing to loss of orbital fat. There is usually hemiatrophy of the tongue on the same side. Disturbance of sensation and muscle twitching may precede or accompany the atrophy. In a majority of the cases the atrophy has been confined to one side of the face, but there are instances on record in which the disease was bilateral, and a few cases in which there were areas of atrophy on the back and on the arm of the same side. The disease is rare; only about 100 cases are in the literature (Möbius).

Of the autopsies, Mendel's alone is satisfactory. There was the terminal stage of an interstitial neuritis in all the branches of the trigeminus, from its origin to the periphery, most marked in the superior maxillary branch.

The disease is recognized at a glance. The facial asymmetry associated with congenital wryneck must not be confounded with progressive facial hemiatrophy. Other conditions to be distinguished are: Facial atrophy in anterior polio-myelitis, and more rarely in the hemiplegia of infants and adults; the atrophy following nuclear lesions and sympathetic nerve paralysis; acquired facial hemihypertrophy, such as in the case recorded by D. W. Montgomery, which may by contrast give to the other side an atrophic appearance; and, lastly, scleroderma (a closely related affection), if confined to one side of the face. The precise nature of the disease is still doubtful, but it is a suggestive fact that in many of the cases the atrophy has followed the acute infections. It is incurable.

V. ACROMEGALY.

Definition.—A dystrophy characterized by abnormal processes of growth, chiefly in the bones of the face and extremities.

The term was introduced by Marie, and signifies large extremities.

Etiology.—It occurs rather more frequently in women. The affection usually begins about the twenty-fifth year, though in some instances as late as the fortieth. Rheumatism, syphilis, and the specific fevers have preceded the development of the disease, but probably have no special connection with it. In this country many cases have now been reported.

Symptoms.—In a well-marked case the disease presents most characteristic features. The hands and feet are greatly enlarged, but are not deformed, and can be used freely. The hypertrophy is general, involving all the tissues, and gives a curious spade-like character to the hands. The lines on the palms are much deepened. The wrists may be enlarged, but the arms are rarely affected. The feet are involved like the hands and are uniformly enlarged. The big toe, however, may be much larger in proportion. The nails are usually broad and large, but there is no curving, and the terminal phalanges are not bulbous. The head increases in volume, but not as much in proportion as the face, which becomes much elongated and enlarged in consequence of the increase in the size of the superior and inferior maxillary bones. The latter in particular increases greatly in size, and often projects below the upper jaw. The alveolar processes are widened and the teeth separated. The soft parts also increase in size, and the nostrils are large and broad. The eyelids are sometimes greatly thickened, and the ears enormously hypertrophied. The tongue in some instances becomes greatly enlarged. Late in the disease the spine may be affected and the back bowed—kyphosis. The bones of the thorax may slowly and progressively enlarge. With this gradual increase in size the skin of the hands and face may appear normal. Sometimes it is slightly altered in color, coarse, or flabby, but it has not the dry, harsh appearance of the skin in myxœdema. The muscles are sometimes wasted. Changes in the thyroid

have been found, but are not constant. The gland has been normal in some, atrophied in others, and in a third group of cases enlarged. Erb, who has made an elaborate study of the disease, has noticed an area of dulness over the manubrium sterni, which he thought possibly due to the persistence or enlargement of the thymus. Headache is not uncommon. Somnolence has been noted in many cases. Menstrual disturbance may occur early, and there may be suppression. *Ocular symptoms* are common. Hertel has analyzed 175 recorded cases, 92 of which presented eye complications. In three fourths of these the optic nerves were affected—usually atrophy, rarely neuritis. Bitemporal hemianopia is often an early sign. The disease may persist for fifteen, twenty, or more years.

Pathological Anatomy.—Furnival has recently analyzed the recorded autopsies, 34 in number. Changes in the pituitary gland were found in all, and in the majority there was hypertrophy or tumor. In 24 cases in which it was examined the thyroid was normal in 5, hypertrophied in one half; the thymus in 17 examined was absent in 7, hypertrophied in 3, and persistent in 7. In Osborne's case the heart was enormous, weighing 2 pounds 9 ounces.

Owing to the remarkable changes in the pituitary gland in acromegaly, it has been suggested that the disease is a nutritional disturbance analogous to myxœdema, and caused directly by disturbance in the function of this organ. The evidence from comparative anatomy and embryology shows that the pituitary body is a very "complex organ, consisting of an anterior secreting glandular organ; a water-vascular duct; a posterior, sensitive, nervous lobe, of which the last two—namely, the duct and the nervous lobe—were morphologically well developed and functioned in ancestral vertebrates, but have become obliterated and atrophied in structure and function forever above larval acraniates" (Andriezen, British Medical Journal, 1894, i). The pituitary body continues active, but the duct is obliterated "and the gland changed into a ductless gland; the secretion becomes an 'internal secretion,'" which is absorbed by the lymphatics. The extraordinary frequency with which the pituitary is involved in this disease lends weight to the view that it is, in the words of Woods Hutchinson, the growth centre, or at any rate the proportion regulator of the skeleton.

It has been suggested by Massalongo and others that gigantism and acromegaly are one and the same disease, both due to the superfunction of the pituitary gland. Certain persons exhibited as giants, or who have been "strong men" and wrestlers, have become acromegalic, and the skulls of some notable giants show enormous enlargement of the sella turcica.

There is a congenital progressive hypertrophy of one extremity or of a part of it or of one side of the body—the so-called *giant growth*, which does not appear to have any connection with acromegaly.

The treatment does not appear to have any influence upon the progress of the disease. The thyroid extract has been tried in many cases, without, so far as my personal experience goes, any benefit. Extract of the pituitary gland has also been used. The lung extract has been employed in some cases of pulmonary osteo-arthropathy. In a case of Caton's, of Liverpool, an unsuccessful attempt was made to extirpate the pituitary body.

Osteitis Deformans (*Paget's Disease*).

In this remarkable affection the shafts of the long bones are chiefly involved, and in the head the bones of the cranium, but not those of the face. It is a rare disease. The bones enlarge and soften, and those bearing weight become unnaturally curved and misshapen. At its commencement, and sometimes through all its course, it is attended with pains in the affected bones.

The bone structure shows a mixture of rarefying osteitis, with the Haversian canals large and irregular, and of formative osteitis, with certain Haversian canals narrowed and lamellæ of recent formation.

There is an intimate relation between osteitis deformans and the formation of malignant tumors. Of 8 cases traced to the end, 5 died with cancer or sarcoma.

About 60 cases have now been recorded, most of them in England. Seven have been reported in America. The most typical case is one reported by Watson in the Johns Hopkins Hospital Bulletin for June, 1898. I saw the man first in July, 1897. At the age of forty-two he was strong and healthy, measuring 5 feet $11\frac{3}{4}$ inches in height. His tibiæ began to enlarge and bow forward and outward, the thoracic spine to curve, and the cranial bones to enlarge. This has steadily progressed. He is now sixty-two years of age. At present, owing to the bowing of the spine and lower extremities, his height is about 5 feet $2\frac{1}{2}$ inches, or $9\frac{1}{4}$ inches less than formerly. The circumference of his head has increased $3\frac{1}{16}$ inches. His thorax is almost perfectly quadrilateral. His intellect is unimpaired, and his general health is fairly good (Watson).

As Marie states, in Paget's disease the face is triangular with the base upward; in acromegaly it is ovoid or egg-shaped with the large end downward; while in myxœdema it is round and full-moon-shaped.

Concerning the etiology of the disease, absolutely nothing is known. No method of treatment has had the slightest influence upon its progress.

Hypertrophic Pulmonary Arthropathy.

Marie has given the name *hypertrophic pulmonary osteo-arthropathy* to a remarkable disorder, first recognized by Bamberger, characterized by enlargement of the hands and feet, and of the ends of the long bones, chiefly of the lower three fourths of the forearm and legs. Unlike acromegaly, the bones of the skull and of the face are not involved. The terminal phalanges are much spread with both transverse and longitudinal curves; the nails, too, are large and much curved over the ends of the phalanges. Scoliosis and kyphosis are rarely seen. The disease is very chronic, and in nearly all cases has been associated with some long-standing affection of the bronchi, lungs, or pleura (hence the name *pulmonary osteo-arthropathy*), of which sarcoma, chronic bronchitis, chronic tuberculosis, and empyema have been the most frequent. There are several instances in which the affection has developed in the subjects of syphilis. It occurs usually in adults and in the male sex. Thayer has reported 4 cases from my clinic

Remarkable case (D. Wolfe, Martinsburg-W. Va)
seen 3-29-00. (D. Atkinson has notes). Going
on for five or six years - legs - arms and
head involved.

and has collected 55 typical cases from the literature. Forty-three showed preceding pulmonary affection; of the remaining, 3 followed syphilis, 3 heart-disease, 2 chronic diarrhœa, 1 spinal caries, and 3 unknown causes.

The essential pathology of the disease is very obscure. Marie suggests that the toxines of the pulmonary disease are absorbed into the circulation and exercise an irritant action on the bony and articular structures, causing an ossifying periostitis. Thorburn thinks that it is a chronic tuberculous affection of a large number of bones and joints of a benign type.

Leontiasis Ossea.

Finally, in a remarkable condition known as *leontiasis ossea*, there is hyperostosis of the bones of the cranium, and sometimes those of the face. The description is largely based upon the skulls in museums, but Allen Starr has recently reported an instance in a woman, who presented a slowly progressing increase in the size of the head, face, and neck, the hard and soft tissues both being affected. He has applied to the condition the term *megalo-cephaly*. Putnam states that the disease begins in early life, often as a result of injury. There may be osteophytic growths from the outer or inner tables, which in the latter situation may give the symptoms of tumor.

Micromegaly.

A remarkable condition, the antithesis of acromegaly, has been described by Jonathan Hutchinson and Hastings Gilford (Lancet, 1896, ii, p. 1227) as "mixed premature and immature development." The name *micromegaly* is suggested by Gilford, who describes it as a disease of that part of the nervous system presiding over nutrition, which manifests itself in a smallness and immaturity of some parts or functions and a relative or actual largeness or prematurity of others.

VI. SCLERODERMA.

Definition.—A condition of localized or diffuse induration of the skin.

Lewin and Heller (Die Sclerodermie, Berlin, 1895) have recently collected from the literature 508 cases.

Two forms are recognized: the circumscribed, which corresponds to the keloid of Addison, and to morphœa; and the diffuse, in which large areas are involved.

The disease affects females more frequently than males. The cases occur most commonly at the middle period of life. The *sclerema neonatorum* is a different affection, not to be confounded with it. The disease is more common in this country than statistics indicate. I have reported 8 cases (Jour. of Genito-Urinary and Cutaneous Diseases, January, 1898), since which date I have seen 3 additional cases.

In the *circumscribed form* there are patches, ranging from a few centi-

metres in diameter to the size of the hand or larger, in which the skin has a waxy or dead-white appearance, and to the touch is brawny, hard, and inelastic. Sometimes there is a preliminary hyperæmia of the skin, and subsequently there are changes in color, either areas of pigmentation or of complete atrophy of the pigment—leucoderma. The sensory changes are rarely marked. The secretion of sweat is diminished or entirely abolished. The disease is more common in women than in men, and is situated most frequently about the breasts and neck, sometimes in the course of the nerves. The patches may develop with great rapidity, and may persist for months or years; sometimes they disappear in a few weeks.

The *diffuse form*, though less common, is more serious. It develops first in the extremities or in the face, and the patient notices that the skin is unusually hard and firm, or that there is a sense of stiffness or tension in making accustomed movements. Gradually a diffuse, brawny induration develops and the skin becomes firm and hard, and so united to the subcutaneous tissues that it cannot be picked up or pinched. The skin may look natural, but more commonly is glossy, drier than normal, and unusually smooth. With reference to the localization, in 66 observations the disease was universal; in 203, regions of the trunk were affected; in 193, parts of the head or face; in 287, portions of one or other of the upper extremities; and in 122, portions of the lower extremities. In 80 cases there were disturbances of sensation. The disease may gradually extend and involve the skin of an entire limb. When universal, the face is expressionless, the lips cannot be moved, mastication is hindered, and it may become extremely difficult to feed the patient. The hands become fixed and the fingers immobile, on account of the extreme induration of the skin over the joints. Remarkable vaso-motor disturbances are common, as extreme cyanosis of the hands and legs. In one of my cases tachycardia was present. The disease is chronic, lasting for months or years. There are instances on record of its persistence for more than twenty years. Recovery may occur, or the disease may be arrested. The patients are apt to succumb to pulmonary complaints or to nephritis. Rheumatic troubles have been noticed in some instances; in others, endocarditis. Raynaud's disease may be associated with it, as in 2 cases described by Stephen Mackenzie. I have seen an instance of the diffuse form in which the primary symptoms were those of local asphyxia of the fingers, and in which, with extensive scleroderma of the arms and hands and face, there were cyanosis and swelling of the skin of the feet without any brawny induration. The pigmentation of the skin may be as deep as in Addison's disease, for which cases have been mistaken; scleroderma may occur as a complication of exophthalmic goitre.

The remarkable dystrophy known as *sclerodactylie* belongs to this disorder. There are symmetrical involvements of the fingers, which become deformed, shortened, and atrophied; the skin becomes thickened, of a waxy color, and is sometimes pigmented. Bullæ and ulcerations have been met with in some instances, and a great deformity of the nails. The disease has usually followed exposure, and the patients are much worse during the winter, and are curiously sensitive to cold. There may be

1412 EUTAW PLACE.

Dear Dr Osler:

There is brief reference
to treatment of - [illegible] Sclerema
by superheated dry air" in the Jan
[illegible] 30, 1904 - p. 295.
You will find it - give you the
reference -

Yours

Louis P. Hamburger

changes in the skin of the feet, but the deformity similar to that which occurs in the hand has not been noted. Some of the cases present in addition diffuse sclerodermatous changes of the skin of other parts. In Lewin and Heller's monograph there are 35 cases of isolated sclerodactylism, and 106 cases in which it was combined with scleroderma.

The pathology of the disease is unknown. It is usually regarded as a tropho-neurosis, probably dependent upon changes in the arteries of the skin leading to connective-tissue overgrowth. The thyroid has been found atrophied.

Treatment.—The patients require to be warmly clad and to be guarded against exposure, as they are particularly sensitive to changes in the weather. Warm baths followed by frictions with oil should be systematically used. I have tried the thyroid feeding thoroughly in the diffuse form without success. In a recent case of quite extensive localized scleroderma, after ten weeks' treatment, the patches are softer and the pigmentation much less intense. Salol in 15-grain doses three times a day is stated to have been successful in several cases.

Ainhum.

Here a brief reference may be made to the remarkable trophic lesion described by Da Silva Lima, which is met with in negroes in Brazil, Africa, India, and occasionally in the Southern States. It is confined to the toes, usually the little toe, and begins as a furrow on the line of the digito-plantar fold. This gradually deepens, the end of the toe enlarges, and, usually without inflammation or pain, the toe falls off. The process may last some years. Cases have been reported in this country by Hornaday, Pittman, F. J. Shepherd, and Morrison.

SECTION XI.

DISEASES OF THE MUSCLES.

I. MYOSITIS.

Definition.—Inflammation of the voluntary muscles.

A primary myositis occurs as an acute or subacute affection, and is probably dependent on some unknown infectious agent. Several characteristic cases have been described of late years. That of E. Wagner may be taken as a typical example. A tuberculous but well-built woman entered the hospital, complaining of stiffness in the shoulders and a slight œdema of the back of the hands and forearms. There was paræsthesia, the arms became swollen, the skin tense, and the muscles felt doughy. Gradually the thighs became affected. The disease lasted about three months. The post mortem showed slight pulmonary tuberculosis; all the muscles except the glutei, the calf, and abdominal muscles were stiff and firm, but fragile, and there were serous infiltration, great proliferation of the interstitial tissue, and fatty degeneration. Similar cases have been reported by Unverricht, Hepp, and Jacoby, of New York. In the case reported by Jacoby the muscles were firm, hard, and tender, and there was slight œdema of the skin. The duration of the cases is usually from one to three months, though there are instances in which it has been longer. The swelling and tenderness of the muscles, the œdema, and the pain naturally suggest trichinosis, and indeed Hepp speaks of it as a pseudo-trichinosis. The nature of the disease is unknown. Senator's case presented marked disorders of sensation, and there is a question whether the peripheral nerves are not involved with the muscles. Wagner suggests that some of these cases were examples of acute progressive muscular atrophy. The separation from trichinosis can be made only by removing a portion of the muscle. It has not yet been determined whether the eosinophilia described by Brown is peculiar to the trichinous myositis. There are septic cases in which a diffuse, purulent infiltration of the muscles of different regions occurs. Instances have been reported in which this has been described as the primary affection, the condition of the muscles even passing on to gangrene.

Acute dermato-mycosis.

Ice Xerosis Nottingh.

Myositis Ossificans Progressiva.

Of this rare and remarkable affection 42 cases have been recorded (Matthes). The process begins within the neck or back, usually with swelling of the affected muscles, redness of the skin, and slight fever. After subsiding an induration remains, which becomes progressively harder as the transformation into bone takes place. The disease is very chronic, and ultimately may involve a majority of the skeletal muscles. Nothing is known of the etiology; the condition has often been associated with malformations.

II. MYOTONIA (*Thomsen's Disease*).

Definition.—An infection characterized by tonic cramp of the muscles on attempting voluntary movements. The disease received its name from the physician who first described it, in whose family it has existed for five generations.

While the disease is in a majority of cases hereditary, hence the name myotonia congenita, there are other forms of spasm very similar which may be acquired, and others still which are quite transitory.

Etiology.—All the typical cases have occurred in family groups; a few isolated instances have been described in which similar symptoms have been present. The disease is rare in this country and in England; it seems more common in Germany and in Scandinavia.

Symptoms.—The disease comes on in childhood. It is noticed that on account of the stiffness the children are not able to take part in ordinary games. The peculiarity is noticed only during voluntary movements. The contraction which the patient wills is slowly accomplished; the relaxation which the patient wills is also slow. The contraction often persists for a little time after he has dropped an object which he has picked up. In walking, the start is difficult; one leg is put forward slowly, it halts from stiffness for a second or two, and then after a few steps the legs become limber and he walks without any difficulty. The muscles of the arms and legs are those usually implicated; rarely the facial, ocular, or laryngeal muscles. Emotion and cold aggravate the condition. In some instances there is mental weakness. The sensation and the reflexes are normal. G. M. Hammond has reported three remarkable cases in one family, in which the disease began at the eighth year and was confined entirely to the arms. It was accompanied with some slight mental feebleness. The condition of the muscles is interesting. The patients appear and are muscular, and there is sometimes a definite hypertrophy of the muscles. The force is scarcely proportionate to the size. Erb has described a characteristic reaction of the nerve and muscle to the electrical currents—the so-called myotonic reaction, the chief feature of which is that normally the contractions caused by either current attain their maximum slowly and relax slowly, and vermicular, wave-like contractions pass from the cathode to the anode.

The disease is incurable, but it may be arrested temporarily. The nature of the affection is unknown. In the only autopsy made Dejerine and

Sottas have found hypertrophy of the primitive fibres with multiplication of the nuclei of all the muscles, including the diaphragm, but not the heart. The spinal cord and the nerves were intact. From Jacoby's recent studies it is doubtful whether these changes in the muscles are in any way characteristic or peculiar to the disease. No treatment for the condition is known.

III. PARAMYOCLONUS MULTIPLEX

(*Myoclonia*).

An affection, described by Friedreich, characterized by clonic contractions, chiefly of the muscles of the extremities, occurring either constantly or in paroxysms.

The cases have been chiefly in males, and the disease has followed emotional disturbance, fright, or straining. The contractions are usually bilateral and may vary from fifty to one hundred and fifty in the minute. Occasionally tonic spasms occur. They are not accompanied by any sensory disturbances. In the intervals between the attacks there may be tremors of the muscles. In the severe spasms the movements may be very violent; the body is tossed about, and it is sometimes difficult to keep the patient in bed. Gucci has described a family in which the affection has occurred in three generations.

Weiss has also noted heredity in four generations. According to this author the essential symptoms are continuous or paroxysmal muscular contractions, usually symmetrical and rhythmical, of muscles otherwise normal, which cease during sleep. There are neither psychical nor sensory disturbances. The condition is most common in young males, and is unaffected by treatment. Raymond groups this disease with fibrillary tremors, electric chorea (Henoch), tic non douloureux of the face, and the convulsive tic, under the name of *myoclonies*, believing that it is only one link in a chain of pathological manifestations in the degenerate.

INDEX.

Abasia, 1126, 1136.
Abdominal typhus, 1.
Abducens nerve (see Sixth Nerve), 1048.
Aberrant, thyroid glands, 836; adrenals, 896.
Abortion, in relapsing fever, 55; in small-pox, 65; in syphilis, 251.
Abscess, atheromatous, 771; of brain, 1025; in appendicitis, 522, 526; in glanders, 235; of kidney (pyonephrosis), 886; of liver, 577; of lung, 662; of mediastinum, 686; of parotid gland, 447; of tonsils, 452; perinephric, 900; cerebral, 1025; pyæmic, 163; retro-pharyngeal, 450, 971.
Acanthocephala, 365.
Acardia, 765.
Acarus scabiei, A. folliculorum, 376.
Accentuated aortic second sound, in chronic Bright's disease, 881; in arterio-sclerosis, 774.
Accessory spasm, 1064.
Acephalocysts (see Hydatid Cysts).
Acetonæmia, 426.
Acetone, 424; tests for, 424.
Acetonuria, 864.
Achondroplasia, 841.
Achromatopsia in hysteria, 1116.
Achylia gastrica, 501.
Acne, from bromide of potassium, 1101; rosacea, 382.
Acromegaly, 1142, and gigantism, 1143.
Actinomycosis, 235; pulmonary, 236; cutaneous, 237; cerebral, 237.
Acute bulbar paralysis, 933.
Acute yellow atrophy, 551.
Addison's disease, 828; pill, 254; keloid, 1145.
Adénie, 809.
Adenitis in scarlet fever, 81.
Adenitis, tuberculous, 282, 812; malignant, 191.
Adenoid growths in pharynx, 454.
Adherent pericardium, 696.
Adhesive pylephlebitis, 554.
Adrenals in Addison's disease, 829.
Ægophony, 120, 670.
Afferent system, diseases of, 920.
Ageusia, 1060.
Agoraphobia, 1124.
Agraphia, 992.
Ague, 202.
Ague cake (see Enlarged Spleen), 216.
Ainhum, 1147.
"Air-hunger" in diabetes, 426.
Akinesia algera, 1126.
Akoria, 503.
Albini, nodules of, 767.
Albinism, in leprosy (lepra alba), 341; of the lung, 656.
Albumin, tests for, 856.
Albuminous expectoration in pleurisy, 678.
Albuminuria, 854, and life assurance, 858; cyclic, 855; febrile, 855; functional, 855; in acute Bright's disease, 870; in chronic Bright's disease, 880; in diabetes, 424; in diphtheria, 150; in epilepsy, 1097; in erysipelas, 159; in gout, 415; in pneumonia, 122; in scarlet fever, 79, 80; in typhoid fever, 31; in variola, 64; neurotic, 855; physiological, 855; prognosis in, 858.
Albuminuric retinitis, 1039.
Albuminuric ulceration of the bowels, 513.
Albumosuria, 857.
Alcaptonuria, 865.
Alcohol, effects of, on the digestive system, 381; on the kidneys, 382; on the nervous system, 381; poisonous effects of, 381.
Alcoholic neuritis, 1034.
Alcoholism, 380; acute, 380; and tuberculosis, 382; chronic, 380.
Alexia, 992.
Algid form of malaria, 215.
Allantiasis, 391.
Allocheiria, 924.
Allorrhythmia, 756.
Alloxuric bodies in gout, 409.
Alopecia, in syphilis, 241.
Alternating paralysis (see Crossed Paralysis).
Altitude, effects of high, 346.
Altitude in tuberculosis, 259, 334.
Amaurosis, hysterical, 1040, 1116; toxic, 1040; uræmic, 867, 881; in hæmatemesis, 496.
Amblyopia, 1040; tobacco, 1040; crossed, 1044.
Ambulatory typhoid fever, 14, 34.
Amœba coli (amœba dysenteriæ), 195; in liver abscess, 195, 577; in sputa, 201.
Amœbic dysentery, 195.
Ammoniæmia, 888.

Amnesia, auditory, 990; tactile, 990; visual, 990.
Amphoric breathing, 309, 683.
Amphoric echo, 309.
Amusia, 991.
Amyloid disease, in phthisis, 298; in syphilis, 242; of kidney, 884; of liver, 586.
Amyosthenia, 1125.
Amyotrophic lateral sclerosis, 928.
Anæmia, 789; bothriocephalus, 367; in anchylostomiasis, 360; from Bilharzia, 352; in chlorosis, 792; from gastric atrophy, 469; from hæmorrhage, 789; miner's, 360; brickmaker's, 360; tunnel, 360; from inanition, 791; from lead, 387; idiopathic, 795; in gastric cancer, 489; in gastric ulcer, 482; mountain, 346, 360; in malarial fever, 216; in rheumatism, 170; in syphilis, 240; in typhoid fever, 19; primary or essential, 792; progressive pernicious, 795; secondary or symptomatic, 789; of spinal cord, 966; splenic, 834; toxic, 791.
Anæmic murmurs (see Hæmic Murmurs).
Anæsthesia, dolorosa, 970; in hemiplegia, 1005; in hysteria, 1115; in leprosy, 342; in locomotor ataxia, 924; in Morvan's disease, 975; in railway spine, 1134; in unilateral lesions of the cord, 965.
Analgesia in hysteria, 1115; in Morvan's disease, 975; in syringo-myelia, 975.
Anarthria, 988.
Anasarca (see Dropsy).
Anchylostomiasis, 359.
Anchylostomum duodenale, 359.
Aneurism, 776; arterio-venous, 776, 788; cirsoid, 776; congenital, 788; cylindrical, 776; dissecting, 776; embolic, 776; false, 776; fusiform, 776; mycotic, 776; of the abdominal aorta, 786; of the branches of the abdominal aorta, 787; of the cerebral arteries, 1013; of the cœliac axis, 787; of heart, 753; of the hepatic artery, 787; of the renal artery, 787; of the splenic artery, 787; of the superior mesenteric artery, 787; true, 776.
Aneurism, of thoracic aorta, 777; hæmorrhage in, 781; pain in, 781; Tufnell's treatment of, 784; unilateral sweating in, 782.
Aneurism, verminous, in the horse, 359, 777.
Angina, Ludovici, 450; simplex, 448; suffocativa, 138.
Angina pectoris, 761; pseudo- or hysterical, 763; toxic, 764; vaso-motoria, 764.
Angiocholitis, chronic catarrhal, 557; suppurative and ulcerative, 557.
Angio-neurotic œdema, 1140.
Angio-sclerosis, 773.
Anguillula stercoralis, A. intestinalis, 364.
Animal lymph, 72.
Anisocoria, 1047.
Ankle clonus, in hysterical paraplegia, 941, 1114; in spastic paraplegia, 937; spurious, 1114.
Anorexia nervosa, 503, 1117.
Anosmia, 1038.
Anterior cerebral artery, embolism of, 1011.
Anterior crural nerve, paralysis of, 1072.
Anthomyia canicularis, 378.
Anthracosis, of lungs, 652; of liver, 570.
Anthrax, 224; bacillus, 224; in animals, 224; external, 225; internal, 226.
Anthropophobia, 1124.
Antipneumonic serum, 113.
Antipneumotoxin, 112.
Antitoxine of diphtheria, 141, 155; of pneumonia, 112; of tetanus, 233.
Antityphoid vaccine, 42.
Anuria, 850; complete, from stone, 850; hysterical, 851.
Anus, imperforate, 533.
Aorta, aneurism of, 777; dynamic pulsation of, 782; throbbing, 786, 1126; tuberculosis of, 327.
Aortic incompetency, 709; sudden death in, 712.
Aortic orifice, congenital lesions of, 767.
Aortic stenosis, 715.
Aortic valves, bicuspid condition of, 766; insufficiency of, 709.
Apex pneumonia, 123.
Aphasia, 988; anatomical localization of, 992; ataxic, 991; hemiplegia with, 991; in infantile hemiplegia, 1018; mixed forms of, 992; motor, 991; of conduction, 992; in phthisis, 312; prognosis of, 993; sensory, 989; in typhoid fever, 30; tests for, 993; transient, in migraine, 1103; Wernicke's, 992.
Aphemia (see Aphasia).
Aphonia, hysterical, 1116; in acute laryngitis, 615; in abductor paralysis, 1061; in pericardial effusion, 692.
Aphthæ (see Stomatitis, Aphthous), 441.
Aphthous fever, 347.
Apoplectic *habitus*, 998; stroke, 1001.
Apoplexy, cerebral, 997; ingravescent, 1001; pulmonary, 638.
Appendicitis, 519; obliterans, 520; infective, 521; perforative, 520; relapsing, 527; ulcerative, 521.
Appendicular colic, 520, 524.
Appendix vermiformis, situation of, 519; perforation of, in typhoid fever, 10; fæcal concretions in, 519; foreign bodies in, 520; necrosis and sloughing of, 521.
Apraxia, 989.
Aprosexia, 454, 456.
Arachnida, parasitic, 375.
Arachnitis (see Meningitis), 954.
Aran-Duchenne type of muscular atrophy, 929, 941; in lead-poisoning, 388.

Arch of aorta, aneurism of, 778.
Arcus senilis, 750.
Argyll Robertson pupil, 1047; in ataxia, 922.
Arithmomania, 1089.
Arm, peripheral paralysis of (see PARALYSIS OF BRACHIAL PLEXUS).
Arrhythmia, 756.
Arsenical neuritis, 1035.
Arsenical pigmentation, 390; in chorea, 1085.
Arsenical poisoning, 390; paralysis in, 391.
Arteries, diseases of, 770; calcification of, 770; degeneration of, 770; fatty, 770; hyaline, 770; tuberculosis of, 327.
Arterio-capillary fibrosis, 770.
Arterio-sclerosis, 770; diffuse, 772; in lead-poisoning, 389; in migraine, 1103; nodular form, 771; in phthisis, 316; senile form, 772.
Arteritis in typhoid fever, 12, 21.
Arteritis, syphilitic, 250.
Arthralgia from lead, 389.
Arthritides, post-febrile, 165; in gout, 414.
Arthritis, 173; acute, in infants, 173; gonorrhœal, 256; in acute myelitis, 977; in cerebro-spinal meningitis, 106; in chorea, 1080; in dengue, 100; in dysentery, 200; in hæmophilia, 820; in small-pox, 65; in tabes dorsalis, 935; multiple secondary, 173; in purpura, 815; rheumatoid, 399; in scarlet fever, 80; septic, 173.
Arthritis deformans, 399; as a chronic infection, 400; in children, 403; chronic form, 402; general progressive form, 402; *Heberden's* nodes in, 401; partial or mono-articular form, 403.
Arthropathies in tabes, 925.
Arthropathy, hypertrophic pulmonary, 1144.
Ascariasis, 352.
Ascaris lumbricoides, 352.
Ascites, 605, 609; chylous, 607; from cancerous peritonitis, 605; from cirrhosis of the liver, 572; from syphilis of the liver, 249; in cancer of the liver, 584; in tuberculous peritonitis, 287; physical signs of, 606; treatment of, 607.
Ascitic fluid, chylous, 607; serous, 607; hæmorrhagic, 607.
Aspergillus in lung, 302.
Asphyxia, local, 1137; death by, in phthisis, 317.
Aspiration, *Bowditch's* conclusions on, 677; in empyema, 678; in pericardial effusion, 695; in pleuritic effusion, 677.
Aspiration pneumonia, 642.
Astasia-abasia, 1126, 1136.
Asthenic bulbar paralysis, 947.
Asthenopia, nervous, 1124.
Asthma, bronchial, 628; nasal affections in, 629; sputum in, 630; cardiac, 628; hay, 612; *Leyden's* crystals in, 631; renal, 867; thymic, 618, 844.
Astrophobia, 1124.
Atavism, in hæmophilia, 819; in gout, 408.
Ataxia, cerebellar, 987; cerebellar-heredo, 950; hereditary, 949; in progressive paresis, 962; locomotor, 920; after small-pox, 64.
Ataxic gait, 923.
Ataxic paraplegia, 948.
Atelectasis, pulmonary, 642.
Atheroma (see ARTERIO-SCLEROSIS and PHLEBO-SCLEROSIS).
Athetosis, 1019; bilateral or double, 939.
Athlete's heart, 710.
Athyrea, 837, 840.
Atmospheric pressure, effects of, 969.
Atremia, 1126.
Atrophic cirrhosis, 571.
Atrophy, acute yellow, of liver, 551; of brain, diffuse, in general paresis, 961; of brain, unilateral, 1017; of muscles, various forms of, 935; progressive muscular, of central origin, 928; unilateral, of face, 1141.
Attitude, in pseudo-hypertrophic muscular paralysis, 934; in paralysis agitans, 1077.
Auditory centre, affections of, 1056; nerve, diseases of, 1056; vertigo, 1058.
Aura, forms of, in epilepsy, 1095.
Auto-infection in tuberculosis, 273.
Automatism, in *petit mal*, 1097; in cerebral syphilis, 246.
Autumnal fever, 3.
Avian tuberculosis, 258.

Baccelli's sign, 670, 672.
Bacillus, anthracis, 224; of cholera, 175.
Bacillus coli communis—distinction from typhoid bacillus, 4; in bile-passages, 558; in fæces of sucklings, 508; in fat necrosis with colitis, 591; in peritonitis, 597.
Bacillus diphtheriæ, 140, 451; value of, in diagnosis, 151.
Bacillus gas (B. aerogenes capsulatus) in peritonitis, 597; in pneumaturia, 864; in pneumopericardium, 698.
Bacillus icteroides, 183.
Bacillus, Klebs-Loeffler, 140; toxine of, 141.
Bacillus, of glanders, 233; of influenza, 96; of smegma, 238; in whooping-cough, 92; of leprosy, 340; of plague, 190; of syphilis, 238; of tetanus, 231; pyocyaneus, 163; strepto-, in typhus fever, 50.
Bacillus mallei, 233.
Bacillus pestis, 190.
Bacillus pneumoniæ, 111.
Bacillus proteus fluorescens, 344.
Bacillus smegma, 238.
Bacillus tuberculosis, 259, 666; diagnostic value of, 313; distribution of, 261; in sputum, 300; methods of detection, 301; outside the body, 261; products of growth of, 260.
Bacillus typhi abdominalis, 3.
Bacteræmia, 161.

Bacteria, proteus group in diarrhœa, 509; relation to diarrhœa, 508.
Bacterium, coli commune (see Bacillus Coli Communis); lactis aerogenes, 508.
Balanitis in diabetes, 425.
Balantidium coli, 351.
Ball-thrombus in left auricle, 723.
Ball-valve stone in common duct, 566.
Banting's method in obesity, 439.
"Barben cholera," 394.
Barking cough of puberty, 1117.
Barlow's disease, 825.
Barrel-shaped chest in emphysema, 658; in enlarged tonsils, 455.
Basedow's disease, 836.
Basilar artery, embolism and thrombosis of, 1010.
Baths, cold, in typhoid fever, 44; in hyperpyrexia of rheumatism, 175; in scarlet fever, 84.
Batophobia, 1124.
Beaded ribs in rickets, 436.
Bed-bug, 377.
Bednar's aphthæ, 443.
Bed-sores, acute, in myelitis, 977, 978; in typhoid fever, 19.
Beer-drinkers, heart disease in, 745.
Bell's (*Luther*) mania, 1075.
Bell's palsy, 1051.
β-oxy-butyric acid, 426, 865.
Beri-beri, 220; forms of, 222.
Besoin de respirer, 346.
Biernacki's symptom, 963.
"Big-jaw" in cattle, 235.
Bile coloring matter, tests for, 549.
Bile-ducts, acute catarrh of, 555; ascarides in, 560; cancer of, 559; congenital obliteration of, 561; stenosis of, 560.
Bile-passages, diseases of, 555.
Bilharzia hæmatobia, 352.
Biliary cirrhosis of liver, 570.
Biliary colic, 563.
Biliary fistulæ, 567.
Bilious remittent fever, 213.
Birth palsies, 938.
Black death, 190.
Black spit of miners, 654.
Black vomit, 186; in dengue, 100.
Bladder, paralysis of, in locomotor ataxia, 922; care of, in myelitis, 979; hypertrophy of, in diabetes insipidus, 432; tuberculosis of, 325.
"Bleeders," 819.
Bleeding, in arterio-sclerosis, 775; in cerebral hæmorrhage, 1012; in emphysema, 659; in heart-disease, 731; in pneumonia, 135; in sun-stroke, 398; in yellow fever, 188.
Blepharospasm, 1055.
Blindness (see Amaurosis).
Blood and ductless glands, diseases of, 789.
Blood, characters of, in anæmia, 789; in cancer of the stomach, 489; in chlorosis, 792; in cholera, 178; in diabetes, 421; in gout, 410; in hæmophilia, 820; in leukæmia, 806; in pernicious anæmia, 797; in pseudo-leukæmia, Hodgkin's disease, 812; in purpura, 814; in secondary anæmia, 790; in typhoid fever, 19.
Blood serum therapy in diphtheria, 155; in pneumonia, 112; in tetanus, 233; in typhoid fever, 47.
Blood-vessels of liver, affections of, 553.
"Blue disease," 768.
Blue line on gums in lead-poisoning, 387.
Boils, in diabetes, 425; after typhoid fever, 19; after small-pox, 65.
Bones, lesions of, in acromegaly, 1142; in congenital syphilis, 244; in leukæmia, 808; in rickets, 434; in typhoid fever, 32.
Borborygmi, 498, 507.
Bothriocephalus latus, 366; anæmia, 367.
Botulism, 391.
Botyroid liver in syphilis, 249.
Bovine tuberculosis, 258.
Bowel, affections of (see Intestines); acute obstruction of, 534; infarction of, 546.
Brachial plexus, affections of, 1069.
Brachycardia (Bradycardia), 759; in typhoid fever, 21.
Brain, diffuse and focal diseases of, 979; abscess of, 1025; abscess of, in congenital heart-disease, 768; affections of blood-vessels of, 994; anæmia of, 995; atrophy and sclerosis of, 1017; congestion of, 994; cysts in, 1021; echinococcus of, 374; hæmorrhage into, 997; syphilis of, 244, 1020; glioma of, 1020; hyperæmia of, 994; inflammation of, 1024; œdema of, 997; porencephalus of, 1017.
Brain-murmur in rickets, 437.
Brain, sclerosis of, 957; diffuse, 958; insular, 959; miliary, 958; tuberous, 959.
Brain, softening of, red, yellow, and white, 1009.
Brain, tubercle of, 321, 1020.
Brain, tumors of, 1020; medical treatment of, 1024; surgical treatment of, 1024; symptoms, general and localizing, 1021.
Brand's method in typhoid fever, 43.
Breakbone fever (see Dengue), 99.
Breast-pang, 761.
Breath, odor of, in diabetic coma, 426; foul, in scurvy, 823; fœtid, in enlarged tonsils, 456.
Breathing (see Respiration); mouth, 454.
Bremer's blood test in diabetes, 427.
Brick-maker's anæmia, 360.
Bright's disease, acute, 869; interstitial form of, 870.
Bright's disease, chronic, 874; interstitial form of, 877; causes of, 877; cardio-vascular changes in, 880; hereditary influences in, 877; parenchymatous form of, 875.
Broadbent's sign, 696.

"Broken-winded," 742.
Bromatotoxismus, 391.
Bromism, 1100.
Bronchi, casts of, 633; diseases of, 621.
Bronchial asthma, 628.
Bronchial catarrh (bronchitis), 621.
Bronchial glands, tuberculosis of, 283; enlargement in whooping-cough, 94, 684; suppuration in, 684.
Bronchiectasis, 626; abscess of brain in, 627; congenital, 626; cylindrical, 626; rheumatoid affections in, 627; saccular, 626; sputum in, 627; universalis, 626.
Bronchiolitis exudativa, 628.
Bronchitis, 621; acute, 621; capillary, 641.
Bronchitis, chronic, 623.
Bronchitis, fibrinous, 632.
Bronchitis in measles, 87; in small-pox, 64; in typhoid fever, 27; putrid, 625.
Bronchocele (see Goitre), 855.
Bronchophony, in pneumonia, 120.
Broncho-pneumonia, acute, 641; chronic, 649; acute tuberculous, 292.
Bronchorrhagia, 637.
Bronchorrhœa, 624; serous, 624.
Bronze-skin, in phthiriasis, 377; in Addison's disease, 830; in Basedow's disease, 859; in diabetes, 425; in Hodgkin's disease, 812.
Brown atrophy of heart, 750.
Brown induration of lung, 635.
Brown-Séquard's paralysis, 965.
Bruit, d'airain, 683; *de cuir neuf*, 690; *de diable*, 794; *de pot fêlé* (see Cracked-pot Sound), 309; *de souffle*, 703; œsophageal, 460.
Bubo, parotid (see also Parotitis), 447.
Bubonic plague, 189.
Buccal psoriasis, 446.
Buhl's disease, 818.
Bulbar paralysis, 928, 932; acute, 933; asthenic form, 947; of cerebral origin, 932; progressive, 928.
Bulimia, 423, 502.

Cachexia, in cancer of the stomach, 489; malarial, 208, 216; periosteal, 825; saturnine, 387; strumipriva, 842; syphilitic, 240.
Caisson disease, 969.
Calcareous concretions, in phthisis, 296; in the tonsils, 456.
Calcareous degeneration, of arteries, 770; of heart, 750.
Calcification, annular, of arteries, 770.
Calcification in tubercle, 271.
Calculi, biliary, 561; "coral," 892; pancreatic, 595; renal, 891; tonsillar, 456; urinary, 891.
Calculous pyelitis, 886.
Camp fever, 49.
Cancer, of bile-passages, 559, 583; of bowel, 533; of brain, 1020; of gall-bladder, 559; green, 809; of kidney, 896; of liver, 582; of lung, 663; of œsophagus, 461; of pancreas, 594; of peritonæum, miliary, 604; of stomach, 486; acute, 493.
Cancrum oris, 444; in measles, 87.
Canities, the result of neuralgia, 1104.
Canned goods, poisoning by, 393.
Capillary pulse, in aortic insufficiency, 714; in neurasthenia, 1126; in phthisis, 311.
Capsule, internal, 982; lesions of, 983.
Caput Medusæ, 606.
Caput quadratum, in rickets, 436.
Carboluria, 865.
Carbuncle in diabetes, 425.
Cardia, spasm of, 499.
Cardiac, compensation, rupture of, 741; disease (see Disease of Heart).
Cardiac murmurs, *hæmic*, in chlorosis, 794; in chorea, 1084; in idiopathic anæmia, 799.
Cardiac murmurs, organic, in aortic insufficiency, 713; in aortic stenosis, 716; in congenital heart affections, 769; in mitral incompetency, 720; in mitral stenosis, 723; in tricuspid valve disease, 726.
Cardiac nerves, neuralgia of, 761.
Cardiac overstrain, 742.
Cardiac septa, anomalies of, 766.
Cardialgia (see Gastralgia).
Cardiocentesis, 755.
Cardio-respiratory murmur, 308.
Cardio-sclerosis, 750.
Cardio-vascular changes in renal disease, 880.
Caries, vertebral, 970.
Carinated abdomen, 278.
Carotid artery, ligature and compression of, in cerebral hæmorrhage, 1012.
Carphologia, 29.
Carpo-pedal spasm, 1111.
Carreau, 288.
Caseation, 271.
Caseous pneumonia, 272.
Casts, blood, of bronchial tubes in hæmoptysis, 638; in fibrinous bronchitis, 633; of pelvis of kidney and ureter, 897.
Casts of urinary tubules, 872; epithelial, 871, 872; fatty, 876; granular, 876, 880; hyaline, 880.
Casts, tube, in acute Bright's disease, 872; in chronic Bright's disease, 876, 880.
Catalepsy in hysteria, 1119.
Cataract, diabetic, 427; after typhoid fever, 30.
Catarrh, acute gastric, 463; autumnal, 612; bronchial, 621; chronic gastric, 466; dry, 625; nasal, 611; simple chronic (nasal), 611; suffocative, 645.
Catarrhal bronchitis, influence of, in tuberculosis, 269.
Catarrhe sec, 625.
Catarrhus æstivus, 612.
Cauda equina, lesions of, 972.
Cavernous breathing, 309.

Cavities, pulmonary, physical signs of, 309; quiescent, 297.
Cayor fly, 379.
Cellulitis of the neck, 450.
Centrum semiovale, lesions of, 981.
Cephalalgia (see HEADACHE).
Cephalic tetanus, 232.
Cephalodynia, 407.
Cercomonas intestinalis, 195, 351; C. hominis, 351.
Cerebellar, ataxia, 950, 987; heredo-ataxia, 950; vertigo, 986.
Cerebellum, tumors of, 986; affections of, 985.
Cerebral arteries, aneurism of, 1013; arteriosclerosis of, 1014; embolism of, 1008; endarteritis of, 1014; syphilitic endarteritis of, 245, 1014; thrombosis of, 1008.
Cerebral cortex, lesions of, 980.
Cerebral hæmorrhage, 997; aneurisms, miliary, in, 998; convulsions in, 1007; forms of, 999.
Cerebral localization, 907.
"Cerebral pneumonia," 122.
"Cerebral rheumatism," 171.
Cerebral sinuses, thrombosis of, 1015.
Cerebral softening, 1008.
Cerebritis (see ENCEPHALITIS), 1024.
Cerebro-spinal meningitis, epidemic, 100; anomalous forms of, 105; complications of, 105; malignant form, 103; ordinary form, 103.
Cervical pachymeningitis, 953.
Cervical plexus, lesions of, 1067.
Cervico-brachial neuralgia, 1105.
Cervico-occipital neuralgia, 1067, 1105.
Cestodes, disease due to, 365; visceral, 368.
Chalicosis, 652.
Chancre, 239.
Charbon, 224.
Charcot's joint, 925.
Charcot-Leyden crystals, 507, 631, 803.
Chattering teeth, 1051.
Cheek, gangrene of, 444.
Cheese, poisoning by, 393.
Chest expansion, diminution of, in Graves' disease, 839.
Cheyne-Stokes breathing, Cheyne's original description of, 751; in apoplexy, 1001; in fatty heart, 751; in sun-stroke, 397; in acute tuberculosis, 275; in uræmia, 867.
Chiasma and tract, affections of, 1041.
Chicken-breast, 436, 455.
Chicken-pox, 74.
Child-crowing, 618.
Children, constipation in, 540; diabetes in, 425; tuberculous broncho-pneumonia in, 292; pneumonia in, 126; tuberculosis of mesenteric glands in, 284, 288; mortality from small-pox in, 65; rheumatism in, 167; typhoid fever in, 35.
Chills (see RIGORS), in typhoid fever, 17.
Chloasma phthisicorum, 313.
Chloro-anæmia in phthisis, 311.
Chloroma, 809.
Chlorosis, 792; and anæmia, sinus thrombosis in, 1015; dilatation of stomach in, 794; Egyptian, 360; fever in, 794; heart symptoms in, 794; menstrual disturbance in, 795; thrombosis in, 794.
Choked disk, 1040.
Cholæmia, 550.
Cholangitis, infective, 566; suppurative, 567, 578; in typhoid fever, 27.
Cholecystectomy, 569; indications for, 569.
Cholecystitis acuta, 564.
Cholecystitis, acute infective, 558.
Cholecystotomy, 569.
Cholelithiasis, 561; in typhoid fever, 27.
Cholera, asiatica, 175; bacillus of, 175; epidemics of, 175; infantum, 509; nostras, 180; sicca, 179; typhoid, 179.
Cholera toxine, 176.
Cholerine, 180.
Cholesteræmia, 550.
Cholesterin in biliary calculi, 563.
Choluria, 865.
Chondrodystrophia fœtalis, 841.
Chorea, acute, 1079; etiology of, 1079; heart symptoms of, 1083; infectious origin of, 1080; in pregnancy, 1080; paralysis in, 1083; rheumatism and, 1079; school-made, 1081.
Chorea, canine, 1080; chronic, 1090.
Chorea, habit or spasm, 1088.
Chorea, *Huntingdon's* or hereditary, 1090.
Chorea, insaniens, 1083, 1085; paralytic form of, 1083; major, 1088; pandemic, 1088; post-hemiplegic, 1019; prehemiplegic, 1001; rhythmic or hysterical, 1091; senile, 1090; spastica, 939, 1086; *Sydenham's*, 1079.
Choroid plexuses, sclerosis of, 1029.
Choroid, tubercles in, 279.
Choroiditis in syphilis, 241.
Chovstek's symptom in tetany, 1110.
Chylangiomata, 547.
Chyle vessels, disorders of, 547.
Chylo-pericardium, 698.
Chyluria, non-parasitic, 859; parasitic, 361.
Cicatricial stenosis of bowel, 533.
Ciliary muscle, paralysis of, 1047.
Ciliata, parasitic, 351.
Cimex lectularius, 377.
Circulatory system, diseases of, 688.
Circumcision, inoculation of tuberculosis by, 264; in hæmophilia, 820.
Circumflex nerve, affections of, 1070.
Cirrhosis, of kidney, 877; of liver, 569; of lung, 649; ventriculi, 467.
Claudication, intermittent, 763.
Claustrophobia, 1124.
Claviceps purpurea, poisoning by, 394.

Clavus hystericus, 1116.
Claw-hand (main en griffe), 930.
Climate, influence of, in asthma, 632: in chronic Bright's disease, 882; in tuberculosis, 333.
Clonus (see Ankle Clonus); jaw, 931.
Clownism in hysteria, 1113.
Cnethocampa, 379.
Cobalt miners, cancer of lung in, 664.
Coccidium oviforme, 349.
Coccydynia, 1106.
Cochin-China diarrhœa, 565.
Cœliac affection in children, 511.
Cog-wheel respiration, 308.
Coin-sound, 683.
Cold pack, method of giving, 84.
Colic, biliary, 563; in appendicitis, 520, 524; in angio-neurotic œdema, 1140; in purpura, 816 lead, 388; mucous, 544; renal, 893.
Colica Pictonum, 386.
Colitis, diphtheritic, 512; mucous, 544; simple ulcerative, 513.
Colles's law, 239.
Colloid cancer, of lung, 663; of peritonæum, 604; of stomach, 487.
Colon, cancer of, 533; dilatation of, 545.
Coloptosis, 543.
Coma, diabetic, 425; epileptic, 1096; from heat-stroke, 396; from muscular exertion, 869; in acute encephalitis, 1025; in acute yellow atrophy, 552; in alcoholic poisoning, 380; in apoplexy, 1001; in cerebral syphilis, 246; in general paresis, 962; in multiple sclerosis, 960; in pernicious malaria, 215; in thrombosis of cerebral sinuses, 1015; in typhoid fever, 29; uræmic, 867.
Coma vigil, 29.
Comatose form of malaria, 215.
Comma bacillus, 175.
Common bile-duct, obstruction of, 566.
Compensation in valve lesions, 708; periods in, 740; rupture of, 741.
Composite portraiture in tuberculosis, 268.
Compressed air disease, 969.
Compression and traction of the bowel, 533.
Compression paraplegia, 970.
Concretions (see Calcareous).
Concussion of spinal cord, 1133.
Confusional insanity, 30.
Congenital heart affections, 765.
Congenital stenosis of pylorus, 494.
Congenital stricture of the bowel, 533.
Congenital syphilis, 242.
Conjugate deviation, in brain tumor, 1023; in apoplexy, 1002; in tuberculous meningitis, 279.
Conjunctiva, diphtheria of, 149.
Consecutive nephritis, 886.
Constipation, 538; in adults, 538; in infants, 540; spasmodic, 539; treatment of, 540.
Constitutional diseases, 399.
Consumption (see Tuberculosis).
Contracted kidneys, 877.
Contracture, hysterical, 1114; in hemiplegia, 1005; of nursing women, 1110.
Contusion pneumonia, 109.
Conus arteriosus, stenosis of, 767.
Conus medullaris, lesions of, 972.
Convalescence, fever of, 16; from typhoid fever, management of, 48.
Convulsions, epileptic, 1096; hysterical, 1112; in acute yellow atrophy, 552; in alcoholism, 380; in aspiration of pleural effusion, 678; in cerebral hæmorrhage, 1001; in cerebral syphilis, 246, 1099; in cerebral tumors, 1021; in chronic Bright's disease, 876.
Convulsions, infantile, 1091; relation to rickets, 438.
Convulsions, in general paralysis, 962; in hepatic colic, 564; in infantile hemiplegia, 1019; in lead-poisoning, 389; in meningitis, 955; in sun-stroke, 397; in typhoid fever, 28; in uræmia, 866; Jacksonian, 1098.
Convulsive tic, 1088, 1089.
Coördination, disturbance of, in tabes, 923.
Copaiba eruption, 88.
Copper test for sugar, 423.
Copræmia, 539, 792.
Coprolalia, 1089.
Cor adiposum, 749.
Cor biloculare, 766.
Cor bovinum, 711.
Cor villosum, 689.
Coronary arteries, in angina pectoris, 762, 763; obliteration of, 747.
Corpora quadrigemina, tumors in, 1023; lesions of, 984.
Corpulence, 439.
Corpus callosum, lesions of, 981.
Corrigan's disease, 709.
Corrigan pulse, 714.
Coryza, acute, 610; fœtida, 612; from the iodides, 254.
Costiveness, 538.
Cough, barking, of puberty, 1117; hysterical, 1116; in acute bronchitis, 622; in chronic bronchitis, 624; in pertussis, 93; in phthisis, 300; during aspiration of pleural effusion, 677; in pneumonia, 118; paroxysmal, in bronchiectasis, 627; paroxysmal, in fibroid phthisis, 314; stomach, 469.
Coup de soleil, 395.
Cow-pox, 68.
Cracked-pot sound, 309.
Cramp, writer's, 1107.
Cramps, in cholera, 180; in gout, 415; in chronic Bright's disease, 882.
Cranio-sclerosis, 437.
Cranio-tabes, relation to congenital syphilis, 436; in rickets, 436.

Craw-craw, 361.
Creophila, 378.
Cretinism, endemic, 840; sporadic, 840.
Cretinoid change, 840.
Crises, gastro-intestinal, in angio-neurotic œdema, 1140; in locomotor ataxia, 924; in purpura, 816.
Crisis, in pneumonia, 117; in relapsing fever; 54; in typhus fever, 51.
Crossed or alternating paralysis, 984, 1004.
Crossed sensory paralysis, 985.
Croup, membranous, 148; spasmodic, 617.
Croupous enteritis, 512.
Croupous pneumonia, 108.
Crura cerebri, lesions of, 983, 1004.
Crutch paralysis, 1070.
Cruveilhier's palsy, 929.
Cry, epileptic, 1096; hydrocephalic, 278; hysterical, 1116; in congenital syphilis, 243.
Cryptogenetic septicæmia, 162.
Curschmann's spirals, 631, 634.
Cyanosis, in acute tuberculosis, 276; in congenital heart-disease, 768; in emphysema, 657.
Cycloplegia, 1047.
Cynanche maligna, 138.
Cynobex hebetica, 1117.
Cystic disease, of kidney, 898; of liver, 584.
Cystic duct, obstruction of, 565.
Cysticercus cellulosæ, 368; ocular, 369; subcutaneous, 369; general, 369; cerebro-spinal, 369.
Cystine calculi, 862, 892.
Cystinuria, 861.
Cystitis, locomotor ataxia, 925; in transverse myelitis, 978; tuberculous, 326.
Cytozoa, 349.
Cysts, chylous, of mesentery, 547; in kidneys, 898; of brain, 1020; porencephalic, 1017; of brain, thrombotic, 1009; pancreatic, 592.

Dacryoadenitis (see LACHRYMAL GLANDS).
Dancing mania, 1088.
Dandy fever (dengue), 99.
Davainea Madagascariensis, 366.
Day-blindness, 1040; in scurvy, 824.
Deaf-mutism after cerebro-spinal fever, 106.
Deafness, in cerebral tumor, 1023; in cerebro-spinal meningitis, 106; in hysteria, 1116; in *Menière's* disease, 1058; in scarlet fever, 81; in tabes dorsalis, 924; nervous, 1057.
Death, modes of, in tuberculosis, 317; sudden, in angina pectoris, 762; in aortic insufficiency, 712; in typhoid fever, 40; in pleural effusion, 671.
Debility, nervous (see NEURASTHENIA), 1122.
Decubitus, acute, 1002; (bed-sores) in transverse myelitis, 978.
Degeneration, reaction of, 914; in neuritis, 1036; in facial paralysis, 1054.
Deglutition, difficult (see DYSPHAGIA).
Deglutition pneumonia, 642.
Delayed resolution in pneumonia, 129.
Delayed sensation in tabes, 924.
Delirium, acute, 1075; acute, in lead-poisoning, 389; cordis, 41, 755, 757; expansive, 962; in acute rheumatism, 171; in pneumonia, 122; in typhoid fever, 29; in typhus fever, 52; tremens, 382.
Deltoid, paralysis of, 1070.
Delusional insanity after pneumonia, 123.
Delusions of grandeur, 962.
Dementia paralytica, 960; alcohol as a factor in, 381; syphilis and, 242, 246, 961.
Demodex folliculorum, 376.
Dengue, 99.
Dentition, in congenital syphilis, 243; in mercurial stomatitis, 445; in rickets, 437.
Dermacentor americanus, 376.
Dermatitis, exfoliative form, 82.
Dermatobia, 378.
Dermatose parasitaire, 361.
Desquamation, in measles, 87; in rubella, 89; in scarlet fever, 79; in small-pox, 62; in typhoid fever, 17.
Deviation, secondary, 1048.
Devonshire colic, 386.
Dextrocardia, 765.
Diabetes insipidus, 432; heredity in, 432; in abdominal tumor, 432; in tuberculous peritonitis, 432.
Diabetes mellitus, 418; acute form, 422; bronzing in, 425; chronic form, 422; coma in, 425; diet in, 428; dietetic form, 422; gangrene in, 425; hereditary influences in, 418; in obesity, 419; in children, 425; lipogenic form, 422; neurotic form, 422; pancreas in, 421; pancreatic form, 422; paraplegia in, 427; perforating ulcer in, 426; theories of, 420; urine in, 423.
Diabetes, phosphatic, 862.
Diabetic, centre in medulla, 419; cirrhosis, 421; coma, 425; phthisis, 421; tabes, 426.
Diacetic acid, 864.
Diaphragm, paralysis of, 1068; degeneration of muscle of, 1068.
Diarrhœa, 505; acute dyspeptic, 509; alba, 511; bacteria in, 508; chronic, treatment of, 514; chylosa, 511; endemic, of hot countries, 364; from anchylostomiasis, 360; in children, treatment of, 516; in cholera, 179; in dysentery, 194, 198, 199; in hysteria, 1117; in phthisis, 311; in typhoid fever, 23; in uræmia, 867; nervous, 506; of Cochin-China, 365; tubular, 544; lienteric, 507.
Diathesis, gouty, 408, 414; hæmorrhagic, 814; lithic acid, 859; tuberculous or scrofulous, 268; uric acid, 860.
Diazo-reaction in typhoid fever, 31.
Dicrotism of pulse in typhoid fever, 13, 20.

dermato-mycosis

Diet, in chronic dyspepsia, 470; in constipation, 540; in convalescence from typhoid fever, 48; in diabetes, 428; in gout, 416; in infantile diarrhœa, 516; in obesity, 439; in scurvy, 822; in tuberculosis, 335; in typhoid fever, 42.
Dietl's crises, 848.
Digestive system, diseases of, 441.
Dioctophyme gigas, 364.
Diphtheria, 138; atypical forms of, 146; of auditory meatus, 149; of conjunctiva, 149; and croup, 144; bacillus of, 140; contagiousness of, 138; hemiplegia in, 150; immunity from, 141; in animals, 139; laryngeal, 148; latent, 147; nephritis in, 150; neuritis in, 151; nasal, 147; pharyngeal, 146; of skin, 149; symptoms of, 146; systemic infection, 147; antitoxine treatment of, 155; of wounds, 149.
Diphtheritic, colitis, 512; membrane, histology of, 144; processes in pneumonia, 115; processes in typhoid fever, 33.
Diphtheritis, 142.
Diphtheroid inflammations, 142.
Diplegia, facial, 1053; in children, 938.
Diplococcus intracellularis meningitidis, 102.
Diplococcus pneumoniæ (micrococcus lanceolatus, pneumococcus), 110; in empyema, 671; in endocarditis, 702; in peritonitis, 597.
Diplopia (see Double Vision), 1049.
Dipsomania, 380.
Dipylidium caninum, 366.
Disinfection in diphtheria, 153; in typhoid fever, 41.
Dissecting aneurism, 776.
Distomiasis, 351.
Distomum lanceolatum, 351; D. buski, 351; D. endemicum, 351; D. perniciosum, 351; D. sinense, 351; D. felineum, 351; D. westermanni, 638, 352.
Dittrich's plugs, 625.
Diuresis, 432.
Diver's paralysis, 969.
Diverticula of œsophagus, 462.
Dochmius duodenalis, 359.
Dorsodynia, 407.
Dothiénentérite, 1.
Double heart, 765.
Double vision, 1049; in ataxia, 922; in chronic Bright's disease, 881.
Dracontiasis, 362.
Dracunculus medinensis, 362.
Drainage, and diphtheria, 138; and scarlet fever, 76; and tonsillitis, 451; and typhoid fever, 5.
Dreamy state in epilepsy, 1097.
Dropsy, cardiac, treatment of, 733; in anæmia (œdema), 797; in acute Bright's disease, 870; in aortic insufficiency, 712; in aortic stenosis, 717; in cancer of stomach, 490; in chronic Bright's disease, 876; in mitral insufficiency, 720; in mitral stenosis, 725; in phthisis, 312; in scarlet fever, 80.
Drug-rashes, 83, 814.
Drunkenness, diagnosis from apoplexy, 380, 1007.
Dry Mouth, 447.
Dulness, movable, in pleural effusion, 669; in pneumothorax, 683.
Dumb ague, 217.
Duodenal ulcer, 478; diagnosis of, from gastric, 484.
Duodenum, defect of, 533; ulcer of, 478.
Dura mater, diseases of, 951; hæmatoma of, 952.
Durande's mixture, 568.
Duroziez's murmur, 714.
Dust, diseases due to, 650, 652; tubercle bacilli in, 261.
Dysacusis, 1057.
Dysentery, 193; abscess of liver in, 196, 200; acute catarrhal, 194; amœba coli in, 195; chronic, 199; diphtheritic, 198; treatment of, 201; tropical or amœbic, 195.
Dyspepsia, acute, 463; chronic, 466; nervous, 497.
Dysphagia, hysterical, 1117; in cancer of the œsophagus, 461; in hydrophobia, 228; in œsophagismus, 459; in œsophagitis, 458; in pericardial effusion, 692; in thoracic aneurism, 781; in tuberculous laryngitis, 619.
Dyspnœa, cardiac, treatment of, 733; from aneurism, 781; hysterical, 1116, 1133; in acute tuberculosis, 275; in aortic insufficiency, 712; in cardiac dilatation, 744; in chlorosis, 792; in diabetic coma, 426; in mitral insufficiency, 719; in mitral stenosis, 725; in pericardial effusion, 692; in pneumonia, 117; in phthisis, 304; in œdema of the glottis, 617; in spasmodic laryngitis, 618; uræmic, 867.
Dystrophies, muscular, 933; clinical forms of, 934.

Ear, complications of scarlet fever, 81; affections of, in syphilis, 241, 244; symptoms simulating meningitis, 1027.
Ebstein's method in obesity, 439.
Echinococcus cyst, fluid of, 371, 373.
Echinococcus disease, 370.
Echinococcus, endogenous, 371; exogenous, 371; multilocular, 371, 374.
Echinorhynchus gigas; E. moniliformis, 365.
Echokinesis, 1089.
Echolalia, 1089.
Eclampsia, 1091.
Ectopia cordis, 765.
Eczema, of the tongue, 445; in diabetes, 425; in gout, 414.
Efferent tract, diseases of, 928.
Ehrlich's reaction in typhoid fever, 31.
Elastic tissue in sputum, 301.

Eclampsia nutans, 1089.

Electrical reactions, in exophthalmic goitre, 839; in facial palsy, 1054; in *Landry's* paralysis, 947; in multiple neuritis, 1036; in periodical paralysis, 1136; in polio-myelitis anterior, 944; in *Thomsen's* disease, 1149.
Electrolysis in aneurism, 785.
Elephantiasis, 362.
Emaciation, in anorexia nervosa, 1117; in gastric cancer, 489; in œsophageal cancer, 461; in phthisis, 306.
Embolic abscesses, 164.
Embolism, and aneurism, 776; in chorea, 1082; in typhoid fever, 21; of cerebral arteries, 1008.
Embryocardia, 757; in pneumonia, 120; in typhoid fever, 21.
Emphysema, 654; acute vesicular, 660; atrophic, 659; compensatory, 655; hypertrophic, 655; interstitial, 660.
Emphysema, subcutaneous, after tracheotomy, 687; after aspiration of the pleura, 677; in gastric ulcer, 479; in phthisis, 313; of the mediastinum, 687.
Emprosthotonos in tetanus, 232.
Empyema, 671; bacteriology of, 671; necessitatis, 237, 672, 783; perforation of lung in, 673.
Encephalitis, acute, 1024; meningo-, chronic diffuse, 960; meningo-, fœtal, 938; polio-, of *Strümpell*, 1018; suppurative, 1025; syphilitic, 245.
Encephalopathy, lead, 388.
Enchondroma of lung, 663.
Endarteritis of spinal cord, 967.
Endocarditis, acute, 698; chronic, 705; chronic vegetative, 701; diphtheritic, 699; in chorea, 699, 1084; infectious, 699; in the fœtus, 707, 767; gonorrhœal, 256; in pneumonia, 700; in puerperal fever, 700; in rheumatism, 170, 699; in septicæmia, 700; in typhoid fever, 11; in tuberculosis, 298, 700; malignant, 699; meningitis in, 700; micro-organisms in, 702; mural, 701; recurring, 699; sclerotic, 707; simple or verrucose, 699; syphilitic, 250; ulcerative, 699.
Endophlebitis, 774.
Enteric fever (see Typhoid Fever), 1.
Enteritis, catarrhal, 505; croupous, 512; diphtheritic, 512; in children, 508; phlegmonous, 512; membranous or tubular, 544; ulcerative, 512.
Entero-colitis, acute, 510.
Enteroclysis in cholera, 181.
Enteroliths, 519, 534; as a cause of appendicitis, 519; in sacculi of colon, 539.
Enteroptosis, 541, 847, 1126.
Entozoa (see Animal Parasites), 349.
Eosinophiles in leukæmia, 806; in trichinosis, 357.
Ependymitis, purulent, 277; granular, in general paresis, 961.
Ephemeral fever, 342.
Epidemic hæmoglobinuria, 818.
Epidemic roseola, 89.
Epidemic stomatitis, 347.
Epididymitis (see Orchitis), 251, 326.
Epilepsia, larvata, 1098; nutans, 1066.
Epilepsy, 1093; and alcoholism, 1095; and syphilis, 1095, 1099; heredity in, 1094; in chronic ergotism, 394; in general paresis, 962; in lead-poisoning, 389; in *Raynaud's* disease, 1139; *Jacksonian*, 917, 1098; masked, 1098; post-epileptic symptoms of, 1097; procursive, 1096; reflex, 1095; rotatory, 1096; spinal, 937; surgical treatment of, 1101.
Epileptic fits, stages of, 1096.
Epistaxis, 614; in hæmophilia, 820; in scurvy, 823; in typhoid fever, 27; "renal," 852; vicarious, 614.
Erb-Goldflam's symptom-complex, 947.
Erb's syphilitic spinal paralysis, 940.
Ergotism, 394; convulsive, 394; gangrenous, 394.
Erosion of teeth, 445.
Eructations, nervous, 498.
Eruptions (see Rashes).
Erysipelas, 157; abscess in, 159; after vaccination, 71; facial, 158; in typhoid fever, 33; migrans, 159; puerperal, 157.
Erythema, exudativum, 815; in pellagra, 395; in typhoid fever, 17; in tonsillitis, 452.
Erythromelalgia, 1106, 1139.
Eschar, sloughing, in hemiplegia, 1002.
Eustrongylus gigas, 364.
Exaltation of ideas in general paresis, 962.
Exanthematous typhus, 49.
Exfoliative dermatitis, 82.
Exophthalmic goitre, 836; acute form, 837; diminution of electrical resistance in, 839; pigmentation in, 839; tremor in, 839; urticaria in, 839.
Eye, motor nerves of, paralysis of, 1046; spasm of, 1047.
Eye-strain in migraine, 1102.
Eyes, conjugate deviation of, in brain tumor, 1023; in apoplexy, 1002; in tuberculous meningitis, 279.

Facial, asymmetry, 1064, 1141; diplegia, 1053; hemiatrophy, 1141; hemihypertrophy, 1142; nerve, paralysis of, 1051; paralysis from cold, 1053; paralysis from lesion of trunk of nerve, 1052; paralysis from lesion of cortex, 1052; paralysis, symptoms of, 1053.
Facial spasm, 1055.
Facies, *Hippocratic*, 598; leontina, in leprosy, 341; in mouth-breathers, 455; *Parkinsonian*, 1077; syphilitic, 243; in typhoid fever, 14.
Fæcal, accumulation, 534, 539; concretions, 519, 539; vomiting, 534.
Fæces, bacteria in, 508; in jaundice, 549.
Fallopian tubes, tuberculosis of, 326.
Famine fever (see Relapsing Fever), 53.

Farcy, 233; acute, 234; chronic, 235.
Farcy-buds, 234.
Farre's tubercles, 583.
Fasciola hepatica, 351.
Fat embolism in diabetes, 426.
Fat necrosis, 591; of pancreas, in diabetes, 422.
Fatty degeneration, of arteries, 770; of kidneys, 874; of liver, 585; of the new-born (*Buhl's* disease), 818.
Fatty degeneration of heart, 749; in anæmia, 796.
Fatty stools, 590.
Febricula, 342.
Febris, carnis, 49; recurrens, 53.
Fehling's test for sugar, 423.
Fermentation, test for sugar, 424.
Fetid stomatitis, 442.
Fever, aphthous, 347; in cholera, 179; entero-mesenteric, 1; ephemeral, 342; gastric, 463; glandular, 345; hysterical, 1119; pernicious malarial, 215; in pneumonia, 116; in acute pneumonic phthisis, 290, 293; in acute miliary tuberculosis, 274; in primary multiple neuritis, 1033; in meningitic tuberculosis, 278; in pulmonary tuberculosis, 304; in pyæmia, 164; in pylephlebitis, suppurative, 580; in intermittent fever, 212; in relapsing fever, 54; in remittent fever, 213; in scarlet fever, 77; in septicæmia, 162; in small-pox, 59; in sun stroke, 396; in appendicitis, 524; in secondary syphilis, 240; in typhoid fever, 14; in yellow fever, 185; lung, 108; Malta, 219; Mediterranean, 219; mountain, 346; Neapolitan, 219; putrid malignant, 1; relapsing, 53; rock, 219; ship, 49; slow nervous, 1; splenic, 224; spotted, 49, 101; typhoid, 1; typho-malarial, 39, 214; typhus, 49; undulant, 219; yellow, 182.
Fever, idiopathic intermittent, 163.
Fever, intermittent, in abscess of liver, 579; in ague, 209; in chronic obstruction of bile-passages by gall-stones, 566; in *Hodgkin's* disease, 812; in pyæmia, 164; in pyelitis, 888; in septicæmia, 163; in secondary syphilis, 240; in tuberculosis, 299, 305.
Fibrinous, bronchitis, 632; pneumonia, 108.
Fibroid disease of heart, 747.
Fibrosis, arterio-capillary, 770.
Fièvre, inflammatoire, 397; *typhoïde à forme rénale*, 31.
Fifth nerve, paralysis of, 1050; gustatory branch, 1051; trophic changes in paralysis of, 1050.
Filaria hominis sanguinis, F. bancrofti, F. diurna, F. perstans, 360; F. medinensis, 362.
Filaria loa, F. lentis, F. labialis, F. hominis oris, F. bronchialis, F. immitis, 364.
Filariasis, 360.
Fish, poisoning by, 393.
Fisher's brain murmur, 437.
Fistula in ano in tuberculosis, 315, 320.
Fistula, œsophago-pleuro-cutaneous, 462.
Flatulence, in hysteria, 1117; in nervous dyspepsia, 500; treatment of, 473.
Flea, bite of, 378.
Flint's murmur, 713, 724.
Floating kidney, 542, 846.
Florida fever, 397.
Fluke, bronchial, 352; blood, 352; liver, 351.
Flukes, diseases caused by, 351.
Fœtal heart-rhythm, 757.
Fœtus, endocarditis in, 767; syphilis in, 242; tuberculosis in, 262; white pneumonia of, 247; typhoid fever in, 36.
Folie Brightique, 866.
Follicular tonsillitis, 451.
Food (see DIET).
Food poisoning, 391.
Foot and mouth disease, 347.
Foreign bodies in intestines, 534.
Fourth nerve, 1047; paralysis of, 1047.
Fremitus, vocal, 119, 307; hydatid, 372.
Friction, mediastinal, 687; pericardial, 690; peritoneal, 604; pleural, 808, 670; pleuro-pericardial, 808.
Friedreich's ataxia, 949.
Friedreich's sign in adherent pericardium, 697.
Frontal convolutions, lesions of, 1022.
Frontal sinuses, pentastomes in, 375.
Funnel breast, 307, 455.

Gait, ataxic, 923; in paralysis agitans, 1077; in pseudo-hypertrophic muscular paralysis, 934; in spastic paraplegia, 937; pseudo-tabetic, 426, 1034; *steppage*, in peripheral neuritis, 1034; in diabetic tabes, 426.
Galactotoxismus, 393.
Gall-bladder, diseases of, 555; atrophy of, 566; calcification of, 565; dilatation of, 565; empyema of, 565; forming abdominal tumor, 565; phlegmonous inflammation of, 565.
Gallop-rhythm, 757.
Galloping consumption, 292.
Gall-stone crepitus, 565.
Gall-stones, 561.
Game-birds, poisoning by, 393.
Ganglia, basal, tumors of, 1022.
Gangrene, in diabetes, 425; in ergotism, 394; in pneumonia, 130; in typhoid fever, 12, 22; in typhus, 52; local or symmetrical, 1138; multiple, 1138; of lung, 660; of mouth, 444.
Gangrenous stomatitis, 444.
Garrod's thread test for uric acid, 410.
Gas-bacillus (see BACILLUS AEROGENES CAPSULATUS).
Gastralgia, 501.
Gastrectasis, 474.
Gastric catarrh, acute, 463.
Gastric, crises, 484, 501, 924; fever, 463.
Gastric juice, hyperacidity of, 484, 500; subacidity of, 501.

Gastric spasm, congenital, 495.
Gastric ulcer, 478; clinical forms of, 482.
Gastritis, acute, 463; acute suppurative, 464; chronic, 466; diphtheritic, 465; membranous, 465; mycotic, 466; parasitic, 466; phlegmonous, 464; polyposa, 467; sclerotic, 467; simple, 463; simple chronic, 466; toxic, 465.
Gastrodynia, 501.
Gastrorrhagia, 495.
Gastrotomy, 462.
Gastroxynsis, 500.
General paralysis of the insane (general paresis), 960; diagnosis of from syphilis, 246, 963; influence of syphilis in, 242, 246, 961.
Genito-urinary system, tuberculosis of, 322.
Gentles, 379.
Geographical tongue, 445.
Gerlier's disease, 1059.
German measles, 89.
Giant growth, 1143.
Giants and gigantism, 1143.
Gigantism and acromegaly, 1143.
Gigantoblasts, 799.
Gigantorhynchus gigas, 365.
Gilles de la Tourette's disease, 1089.
Gin-drinker's liver (see Cirrhosis of Liver), 569.
Glanders, 233; acute, 234; chronic, 234; diagnosis from small-pox, 67.
Glandular fever, 345.
Glenard's disease, 541.
Glioma of brain, 1020.
Gliosis, 975.
Globulin in urine, 857.
Globus hystericus, 1112.
Glomerulo-nephritis, 870.
Glosso-labio-laryngeal paralysis, 932.
Glosso-pharyngeal nerve, affections of, 1059.
Glossy skin in arthritis deformans, 402.
Glottis, œdema of, 617; in Bright's disease, 881; in small-pox, 64; in typhoid fever, 11.
Gluteal nerve, affections of, 1072.
Glycogen, formation of, 420.
Glycogenic function of liver, 420.
Glycosuria, 420, 865; gouty, 415; lipogenic, 422.
Gmelin's test, 549.
Goitre, 835; exophthalmic, 836; sudden death in, 836.
Gonorrhœal arthritis, 256; endocarditis, 256; septicæmia and pyæmia, 255.
Gonorrhœal infection, 255; systemic, 255.
Gout, 407; acute, 411; chronic, 413; *Ebstein's* theory of, 409; hereditary influence in, 408; influence of alcohol in, 408; influence of food in, 408; influence of lead in, 408; irregular, 414; nervous theory of, 409; retrocedent or suppressed, 413.
Gouty kidney, 877.
von Graefe's sign, 838.
Grain, poisoning by, 394.
Grandeur, delusions of, 962.
Grand mal, 1094, 1095.
Granular kidney, 877.
Granulomata, infectious, of brain, 1020.
Gravel, renal, 892.
Graves' disease, 836.
Green cancer, 809.
Green-sickness (see Chlorosis), 792.
Green-stick fracture in rickets, 437.
Gregarinidæ, parasitic, 349.
Grinder's rot, 652.
Grippe, la, 95.
Gruebler's tumor, 388.
Guinea-worm disease, 362.
Gull's disease, 841.
Gummata, 239; in acquired syphilis, 241; in congenital syphilis, 244; of brain and spinal cord, 244; of heart, 250; of kidneys, 250; of liver, 248; of lungs, 247; of rectum, 249; of testis, 251; structure of, 239.
Gummatous periarteritis, 250.
Gums, black line on, in miners, 387; blue line on, in lead-poisoning, 387; in scurvy, 823; in stomatitis, 442; red line on, in pulmonary tuberculosis, 311.
Gustatory paralysis, 1051.

Habit spasm, 1088; in mouth-breathers, 456.
Habitus, apoplectic, 998; phthisicus, 268.
Hæmatemesis, 495; causes of, 495; in cirrhosis of liver, 572; diagnosis from hæmoptysis, 497; in enlarged spleen, 216, 495; in scurvy, 823.
Hæmato-chyluria, non-parasitic, 859; parasitic, 361.
Hæmatoma of dura, of brain, 952; of cord, 953; of mesentery, 546.
Hæmatomyelia, 968.
Hæmatoporphyrin, 865.
Hæmatorrhachis, 967.
Hæmatozoa of malaria, 205.
Hæmaturia, 851; endemic, of Egypt, 352; in acute nephritis, 870; in chronic phthisis, 312; in psórospermiasis, 350; in renal calculus, 894; in renal cancer, 897; in tuberculosis of kidney, 325; malaria, 216.
Hæmocytozoa, 205.
Hæmoglobin, reduction of, in chlorosis, 793.
Hæmoglobinæmia, 854.
Hæmoglobinuria, 852; epidemic, in infants, 243; 818, 853; in *Raynaud's* disease, 1138; paroxysmal, 853; toxic, 853.
Hæmo-pericardium, 698.
Hæmo-peritoneum, 588.
Hæmophilia, 819.
Hæmoptysis, causes of, 637; hysterical, 1117; at onset of phthisis, 299; in acute broncho-pneumonic phthisis, 293; in acute miliary tuberculosis, 275; in aneurism, 637, 781; in aortic in-

Head-shaking in infants. 1957

foreign bodies in

Tuberculosis. Lumbar

sufficiency, 712; in arthritic subjects, 638; in bronchiectasis, 627; in cirrhosis of lung, 651; in emphysema, 659; in hysteria, 1117; in mitral insufficiency, 720; in mitral stenosis, 725; in pneumonia, 118; in pulmonary gangrene, 662; in scurvy, 823; spurious, 1117; symptoms of, 638; treatment of, 639; in typhoid fever, 28; relation to tuberculosis, 637; parasitic, 352; periodic, 637; vicarious, 637.
Hæmorrhage, broncho-pulmonary, 637; cerebral, 997; from mesentery, 546; from the stomach, 495; in acute yellow atrophy, 552; in anæmia, 799; in cirrhosis of the liver, 572; in contracted kidney, 882; in gastric cancer, 490; in gastric ulcer, 481; in hæmophilia, 820; in hysteria, 1117, 1118; in intussusception, 537; in leukæmia, 805; in malaria, 216; in nephrolithiasis, 894; in the new-born, 818; in purpura hæmorrhagica, 816; in scarlet fever, 79; in scurvy, 823; in small-pox, 62; in splenic enlargement, 216, 495; into pancreas, 588; into spinal cord, 968; into spinal membranes, 967; into tuberculous pyelitis, 325; in tuberculosis of bowels, 319; into ventricles of brain, 999; in typhoid fever, 10, 24; in yellow fever, 186; pulmonary, 302, 637.
Hæmorrhagic diathesis, 814.
Hæmorrhagic diseases of the new-born, 818.
Hæmorrhagic typhoid fever, 35.
Hæmothorax, 674.
Hair tumors in stomach, 494.
Hallucinations in hysteria, 1119.
Harrison's groove in rickets, 436; in enlarged tonsils, 455.
Harvest-bug, 376.
Hay-asthma (hay-fever), 612.
Haygarth's nodosities, 401.
Headache, from cerebral tumor, 1021; in cerebral syphilis, 246; in mouth-breathers, 456; in typhoid fever, 12, 14, 28; in uræmia, 867; sick, 1102.
Head-cheese, poisoning by, 391.
Heart, diseases of, 698; diseases of, *Oertel's* treatment of, 752; amyloid degeneration of, 750; aneurism of, 753; athlete's, 710; brown atrophy of, 750; calcareous degeneration of, 750; congenital affections of, 765; dilatation of, 741; displacement in pleuritic effusion, 667; displacement in pneumothorax, 682; fatty disease of, 749; fragmentation of fibres of, 748; hydatids of, 754; hypertrophy of, 735; hypertrophy of, in Bright's disease, 880; in exophthalmic goitre, 838; irritable, 745, 756; new growths in, 754; neuroses of, 755; palpitation of, 755; parenchymatous degeneration of, 748; rupture of, 753; valvular diseases of, 707; wounds of, 754.
Heart-muscle in fevers, 748.
Heart-sounds, weakness of, 744; increased loudness of, 739; audible at distance, 724, 838.
Heart-valves, congenital anomalies and lesions of, 766; rupture of, 711.
Heat, exhaustion, 395; stroke, 395.
Heberden's nodes, 401.
Hectic fever, 306.
Heel, painful, 1106.
Heller's test, 856.
Helminthiasis (see Animal Parasites), 349.
Hemeralopia, 1040; in scurvy, 824.
Hemialbumose, 857.
Hemianæsthesia, in cerebral hæmorrhage, 1005; in hysteria, 1115; in lesions of internal capsule, 983; in unilateral cord lesions, 965.
Hemianopia, heteronymous, 1042; homonymous, 1042; in migraine, 1102; lateral, 1042; nasal, 1042; significance of, 1045; temporal, 1042.
Hemicrania, 1102.
Hemiopic pupillary inaction, 1044.
Hemiplegia, 1002; crossed, 984, 1004.
Hemiplegia, infantile, 1017; aphasia in, 1018; in diphtheria, 150; epilepsy in, 1019; in hysteria, 1114; mental defects in, 1018; post-hemiplegic movements in, 1019; spastica cerebralis, 1018; in typhoid fever, 30.
Hémiplégie flasque, 1006.
Henoch's purpura, 816.
Hepatic, abscess, 577; artery, enlargement of, 555; colic, 563; intermittent fever, 566; vein, affections of, 555.
Hepatitis, diffuse syphilitic, 248; interstitial (see Cirrhosis), 569; suppurative, 577.
Hepatization, of lung, 113; white, of fœtus, 247.
Hereditary form of œdema, 1141.
Heredity, in Bright's disease, 877; in diabetes insipidus, 432; in *Friedreich's* ataxia, 949; in gout, 408; in hæmophilia, 819; in paramyoclonus multiplex, 1150; in spastic paraplegia, 940; in syphilis, 238; in tuberculosis, 262.
Herpes, in trifacial neuralgia, 1105; in cerebro-spinal meningitis, 104; in febricula, 343; in malaria, 212; in pneumonia, 122; zoster, 1106.
Hiccough, 1068; causes of, 1068; treatment of, 1069; hysterical, 1116.
High-tension pulse, characters of, 774, 880.
Hippocratic facies, 598; fingers, 313; succussion, 683.
Hippus, 1102.
Hodgkin's disease, 809; intermittent fever in, 812.
Homalomyia scalaris, 378.
Horn-pox, 63.
Hospital fever, 49.
Huntingdon's chorea, 1090.
Husband and wife, diabetes in, 418; tuberculosis in, 266.
Hutchinson's teeth, 243.
Hyaline casts in urine, 871, 876, 880.

Hybrid measles, 89.
Hydatid disease (see Echinococcus).
Hydatid thrill or fremitus, 372.
Hydrarthrosis, chronic, 257; intermittent, 1118.
"Hydrencephaloid condition," 510, 996.
Hydriatic treatment (see Hydrotherapy).
Hydrocephalus, 1028; acquired chronic, 1029; acute, 276, 1028; angio-neurotic, 1028; chronic, after cerebro-spinal meningitis, 106; congenital, 1029; idiopathic internal, 1028; spurious, 510.
Hydromyelus, 953, 975.
Hydronephrosis, 889; congenital, 889; intermittent, 848, 890.
Hydropericardium, 697.
Hydroperitonæum, 605.
Hydrophobia, 227.
Hydro-pneumothorax, 681.
Hydrops vesicæ felleæ, 565.
Hydrothorax, 680.
Hymenolepsis diminuta; H. nana, 366.
Hyperacidity, 500.
Hyperacusis, 1057.
Hyperæsthesia, in ataxia, 924; in hysteria, 1115; in rickets, 435; in unilateral cord lesions, 965.
Hyperchlorhydria, 500.
Hyperosmia, 1038.
Hyperpyrexia, hysterical, 1120; in rheumatic fever, 170; in scarlet fever, 78; in sun-stroke, 396; in tetanus, 232.
Hyperthyrea, 837.
Hyperthyroidism, 836.
Hypertrophic cirrhosis of liver, 574.
Hypnotism in hysteria, 1121.
Hypodermic syringe in diagnosis of pleural effusion, 675.
Hypoglossal nerve, diseases of, 1066; paralysis of, 1066; spasm of, 1067.
Hypophysis, enlargement of, 1143.
Hypostatic congestion, of lungs, 635; in typhoid fever, 28.
Hypotonia, 924.
Hysteria, 1111; and disseminated sclerosis, 960: contractures and spasms in, 1114; convulsive forms of, 1112; cries in, 1116; disorders of sensation in, 1115; forms of fever in, 1119; hæmoptysis in, 1117; insanity in, 1119; joint affections in, 1118; mental symptoms of, 1118; metabolism in, 1119; metallotherapy in, 1115; non-convulsive forms of, 1113; paralysis in, 1113; special senses in, 1116; stigmata in, 815, 1118; traumatic, 1132; visceral manifestations of, 1116.
Hysterical angina pectoris, 763.
Hystero-epilepsy, 1098, 1113.
Hysterogenic points, 1116.

Ice-cream, poisoning by, 393.
Ice, typhoid bacillus in, 4.
Ichthyosis lingualæ, 446.
Ichthyotoxismus, 393.
Icterus (see Jaundice).
Idiocy in infantile hemiplegia, 1019.
Idiopathic anæmia of *Addison*, 795.
Idiopathic intermittent fever, 163.
Ileo-cæcal region, in typhoid fever, 25; in appendicitis, 525; in primary tuberculosis of bowel, 320.
Ileus (see Strangulation of Bowel), 531.
Imbecility in infantile hemiplegia, 1019.
Imitation in chorea, 1081.
Impetigo, contagious, and ulcerative stomatitis, 442.
Impotence, in diabetes, 427; in locomotor ataxia, 922.
Incarceration of bowel, 531.
Incoördination, of arms, 923; of legs, 923.
Indians, American, chorea in, 1079; consumption in, 259; small-pox among, 56.
Indicanuria, 863.
Indigestion, 463.
Infantile, convulsions, 1091; paralysis, 942; scurvy, 825.
Infantilism, 243, 841.
Infection, definition, 160.
Infectious diseases, 1; of doubtful nature, 342.
Inflation of bowel in intussusception, 538.
Influenza, 95; complications of, 97.
Infusoria, parasitic, 351.
Inhalation pneumonia (see Aspiration Pneumonia), 642.
Inoculation, against small-pox, 56, 63; protective, in cholera, 176; preventive, in hydrophobia, 229; preventive, in plague, 192; preventive, in pneumonia, 112; preventive, in yellow fever, 189: tuberculosis transmitted by, 264.
Insanity, post-febrile, 30; in small-pox, 64.
Insanity, relation of drink to, 381: relations of chronic phthisis to, 312; relation of heart-disease to, 712.
Insects, parasitic, 376.
Insolation, 395.
Insular sclerosis, 959.
Intention tremor (see Volitional Tremor).
Intermittent claudication, 763.
Intermittent fever, 209; forms of (see Fever).
Intermittent hepatic fever, 566.
Intermittent hydrarthrosis, 1118.
Internal capsule, lesions of, 982, 983.
Internal carotid artery, blocking of, 1011.
Intestinal casts, 544; sand, 546.
Intestinal coils, tumor formed by, 288.
Intestinal obstruction, 531.
Intestines, diseases of, 505; actinomycosis of, 236; dilatation of, 545.
Intestines, hæmorrhage from, in typhoid fever, 10, 24; in dysentery, 195, 198; in tuberculosis

x Branch of hyssop needle enlarged, a,

Koplik's sign 87.

in diphtheria,

of bowel, 319; in intussusception of, 537; in ulceration of, 513.
Intestines, infarction of, 546; intussusception of, 532, 537; invagination of, 532; miscellaneous affections of, 544; new growths in, 533.
Intestines, obstruction of, 531, 599; acute, 534; by enteroliths, 534; by foreign bodies, 534; by gall-stones, 534, 568.
Intestines, perforation of, in typhoid fever, 10.
Intestines, primary tuberculosis of, 319; strangulation of, 531, 536; strictures and tumors of, 533; twists and knots in, 533; ulcers of, 512.
Intoxication, definition of, 161.
Intoxications, 380.
Intussusception, 532, 537.
Invagination, 532; post-mortem, 532.
Inverse type of temperature, in acute tuberculosis, 274; in typhoid fever, 16.
Iodide eruptions, 254.
Iridoplegia, 1047; accommodative, 1047; reflex, 1047.
Iritis, syphilitic, 241, 244.
Itch, 376.
Itching, of feet in gout, 415; of eyeballs in gout, 415; of skin in Bright's disease, 882; of skin in jaundice, 549; in diabetes, 425; in exophthalmic goitre, 839.
Ixodes ricinus, 376.

Jacksonian epilepsy, 917, 1098.
Jail fever, 49.
Jaundice, 548; black, 549; catarrhal, 555; choluria in, 549; from cirrhosis of liver, 572, 575; epidemic form of, 344, 550; febrile, 344; from acute yellow atrophy, 551; from cancer of liver, 584; from gall-stones, 564, 566; in influenza, 97; in pneumonia, 125; and purpura, 814, 549; in *Weil's* disease, 344; malignant, 551; of the new-born, 551; obstructive, 548; toxæmic, 550; xanthelasma in, 549; in yellow fever, 185.
Jaw clonus, 931.
Jigger, 378.
Joints (see Arthritis).
Jumpers, 1089.
"June cold," 612.

Kahler's disease, albumosuria in, 857.
Kakke, 221.
Kala-ázar, 203.
Keloid of *Addison*, 1145.
Keratitis, in small-pox, 65; interstitial, of inherited syphilis, 244.
Keratosis follicularis, 350.
Keratosis mucosæ oris, 446.
Kidney, diseases of, 846; amyloid or lardaceous disease of, 884; cancer of, 896; cardiac, 850; circulatory disturbance in, 849; cirrhosis of, 877; congenital cystic, 898; congestion of, 849; contracted, 877; cyanotic induration of, 850; cystic disease of, 898; echinococcus of, 374; floating, 846; fused, 846; gouty, 877; granular, 877; horseshoe, 846; large white, 874; malformations of, 846; movable, 846; palpable, 846.
Kidney, rhabdo-myoma of, 896; sarcoma of, 896; scrofulous, 325, 887; small white kidney, 874; surgical kidney, 887; syphilis of, 250; tuberculosis of, 324; tumors of, 896; unsymmetrical, 846.
Klebs–Loeffler bacillus, 140.
Knee-jerk, loss of, in ataxia, 924; in diphtheria, 151.
Koch treatment of tuberculosis, 335.
Kopftetanus of *Rose*, 232.
Kreotoxismus, 391.
Kubisagari, 1059.

Labyrinthine disease, 1058.
Lachrymal gland in mumps, 91; in Mikulicz's disease, 448.
"Lacing" liver, 587.
Lacunar tonsillitis, 451.
La grippe, 95.
Lamblia intestinalis, 351.
Landry's paralysis, 946.
Laparotomy in typhoid fever, 48.
Larvæ of flies, diseases caused by (myiasis), 378.
Laryngeal crises, 925.
Laryngismus stridulus, 617; from pressure of enlarged thymus, 844.
Laryngitis, acute, catarrhal, 615; chronic, 616; œdematous, 617; spasmodic, 617; syphilitic, 620; tuberculous, 619.
Larynx, diseases of, 615; adductor paralysis of, 1061; anæsthesia of, 1062; hyperæsthesia of, 1062; paralysis of abductors of, 1061; spasm of the muscles of, 1062; unilateral abductor paralysis of, 1061.
Latah, 1089.
Lateral sclerosis, primary, 937; amyotrophic, 928.
Lateritious deposit, 860.
Lathyrism, 394.
Lead, colic, 388; in the urine, 387.
Lead-palsy, 388; localized forms of, 388.
Lead-poisoning, 386; acute, 387; arterio-sclerosis in, 389; cerebral symptoms in, 389; chronic, 387; convulsions from, 389; gouty deposits in, 389; treatment of, 389.
Lead-workers, prevalence of gout in, 408.
Leichen-tubercle, 264.
Leontiasis ossea, 1145.
Lepra alba, 341.
Lepra mutilans, 341.
Leprosy, 338; anæsthetic, 342; bacillus lepræ in, 340; contagiousness of, 340; macular form of, 341; tubercular, 341.

Leptomeningitis, acute cerebro-spinal, 954; chronic, 957; infantum, 957.
Leptothrix in mouth, 236.
Leptus autumnalis, 376.
Leucin, 552.
Leucocytes, relation to uric acid, 409.
Leucocytosis, in anæmia, 791, 799; chlorosis, 794; cerebro-spinal meningitis, 104; diphtheria, 147; empyema, 672; erysipelas, 159; *Hodgkin's* disease, 812; leukæmia, 806; malaria, 217; measles, 88; pyæmia, 165; pneumonia, 120; pleurisy, 670; rheumatic fever, 170; scarlet fever, 82; stomach cancer, 489; in trichinosis, 357; in tuberculosis (acute), 275; in tuberculosis (chronic pulmonary), 311; typhoid fever, 19, 37; in whooping-cough, 94.
Leucoderma, 839, 1146.
Leucomata, 241.
Leukæmia, 802; acute, 808; lymphatic, 808; blood in, 806; congenital, 803; definition of, 802; heredity in, 802; in animals, 803; in pregnancy, 802; morbid anatomy of, 803; myelogenous, 802; prognosis in, 809; pseudo-, 809; spleno-medullary, 805.
Leukoplakia buccalis, 446.
Leyden's crystals, 631, 634.
Lienteric diarrhœa, 507.
Life assurance and albuminuria, 858; and syphilis, 255.
Lightning pains in ataxia, 922.
Lineæ atrophicæ, 19.
Lingual corns, 446.
Lipaciduria, 864.
Lipæmia, 421, 426.
Lipothymia, 599.
Lips, tuberculosis of, 317; chancre of, 238.
Lipuria, 864.
Lithæmia, 859, 860.
Lithic-acid diathesis, 859.
Lithuria, 859.
Little's disease, 938.
Liver, abscess of, 577; actinomycosis of, 236; acute yellow atrophy of, 551; amyloid, 586; anæmia of, 553; angioma of, 584; cardiac, 554; anomalies in form and position of, 587.
Liver, cirrhosis of, 569; alcoholic, 570; ascites in, 572; atrophic, 571; capsular form, 575; in diabetes, 421; fatty, 571; hæmorrhage from stomach in, 572; hypertrophic, 574; syphilitic, 575; in tuberculosis, 320; in children, 570; jaundice in, 572; toxic symptoms in, 573; with cancer, 583.
Liver, cysts of, 584; fatty, 585; gummata of, 248; hepato-phlebotomy in congestion of, 554; hydatids of, 372; hyperæmia of, 553; infarction of, 554; melano-sarcoma of, 583; new growths in, 582; nutmeg, 553; passive congestion of, 553; periodical enlargement of, 553; primary cancer of, 582; psorospermiasis of, 349; pulsation of, 554; sarcoma of, 583; secondary cancer of, 583; syphilis of, 248; tuberculosis of, 320; in typhoid fever, 11, 27.
Liver, diseases of, 548.
Liver dulness, obliteration of, in perforative peritonitis, 26, 598.
Liver, movable, 542, 587.
Living skeletons, 930.
Lobar pneumonia, 108.
Lobstein's cancer, 897.
Localization, cerebral, 907; spinal, 905.
Localized peritonitis, 522, 600.
Lock-jaw, 230.
Lock-spasm, 1108.
Locomotive ataxia, 920; bladder symptoms in, 922; gastric crises in, 924; hemiplegia in, 925; paresis in, 925; rectal crises in, 924; relation of syphilis to, 920; reputed cures of, 927.
Long thoracic nerve, affections of, 1070.
Loose shoulders, 934.
Lucilia macellaria, 378.
Ludwig's angina, 450.
Lues venerea (syphilis), 238.
Lumbago, 406.
Lumbar plexus, lesions of, 1072.
Lumbar puncture of *Quincke*, 107, 956, 1030.
Lung, abscess of, 662; embolic, 662.
Lung, actinomycosis of, 236; albinism of, 656; brown induration of, 635; cancer of, acute, 664; carnification of, 643; cirrhosis of, 649.
Lung, diseases of, 634; stones, 296.
Lung fever, 108.
Lungs, congestion of, 634; hypostatic, 635.
Lungs, echinococcus of, 373.
Lungs, gangrene of, 660; abscess of brain in, 661.
Lungs, new growths in, 663; in cobalt-miners, 664.
Lungs, hæmorrhagic infarction of, 638; œdema of, 636; splenization of, 635, 643; syphilis of, 247; tuberculosis of, 289.
Lupinosis, 394.
Lymphadenitis, general tuberculous, 282; local tuberculous, 282; simple, 684; suppurative, 684.
Lymphadenoma, general, 809.
Lymphatic state, 826.
Lymphatism, 826.
Lymph-scrotum, 362.
Lymph, vaccine, 72.
Lymph vessels, dilatation of, 362.
Lyssa, 227.
Lyssophobia, 230.

Macular syphilides, 240.
Maïdismus, 395.
Main en griffe, 930, 953.
Maize, poisoning by (pellagra), 395.
Malarial cachexia, 208, 216.
Malarial fever, 202; accidental and late lesions

of, 208; æstivo-autumnal, 213; algid form of, 215; comatose form of, 215; continued and remittent form of, 215; description of the paroxysm in, 209; geographical distribution of, 208; hæmorrhagic form of, 216; intermittent, 209; meteorological conditions influencing, 204; pernicious, 207, 215; pneumonia in, 209; quartan, 213; quotidian, 213; season in, 204; specific germ of, 204; telluric conditions influencing, 203; tertian, 212.
Malarial nephritis, 209.
Malignant jaundice, 551.
Malignant purpuric fever, 101.
Malignant pustule, 225.
Mallein, 234.
Malta fever, 219.
Mammary glands, hypertrophy in tuberculosis, 312; tuberculosis of, 327.
Mania a pôtu, 382.
Mania, *Bell's*, 1075.
Marriage, question of, in hæmophilia, 821; in syphilis, 254; in tabes dorsalis, 927; in tuberculosis, 329.
Marrow of bones, in small-pox, 58; in leukæmia, 803; in pernicious anæmia, 797.
Masque des femmes enceintes, 831.
Massai disease, 363.
Mastication, spasm of the muscles of, 1051.
McBurney's tender point, 525.
Measles, 85; complications and sequelæ of, 87; contagiousness of, 85; desquamation in, 87; eruption in, 86; German, 89; period of incubation in, 86.
Measly meat, examination of, 367.
Meat, poisoning by, 391; tuberculous infection by, 267; inspection of, for trichinæ, 355.
Meckel's diverticulum, 532.
Median nerve, affections of, 1071.
Mediastinal friction, 687.
Mediastino-pericarditis, indurative, 687.
Mediastinum, affections of, 684; abscess of, 686; tumors of, 685; cancer of, 685; emphysema of, 687; pleural effusion in, 686; sarcoma of, 685.
Mediterranean fever, 219.
Medulla oblongata, lesions of, 984; tumors of, 1023.
Megalo-cephaly, 1145.
Megalocytes, 798.
Megastrie, 475.
Melæna, in duodenal ulcer, 484; in typhoid fever, 24; in tuberculosis of bowels, 319; neonatorum, 818.
Melano-sarcoma of liver, 583.
Melanuria, 863.
Melasma suprarenale, 831.
Ménière's disease, 1058.
Meningeal hæmorrhage, 999; in birth palsies, 938.
Meninges, affection of, 951.
Meningitis, acute cerebro-spinal, 954; epidemic cerebro-spinal, 100; in erysipelas, 158, 159; in gout, 415; in typhoid fever, 12, 14, 28; ~~ocula-sive, 957~~; posterior, of infants, 957; serosa, 1028; syphilitic, 245; tuberculous, 276.
Meningococcus, 102.
Meningo-encephalitis, chronic diffuse, 960; tuberculous, 277.
Mercurial, tremor, 1079; stomatitis, 444.
Mercyismus, 499.
Mesenteric artery, aneurism of, 546; embolism of, 546; thrombosis of, 546.
Mesenteric glands, tuberculosis of, 283; tuberculous tumors of, 288; in typhoid fever, 10.
Mesenteric veins, diseases of, 547.
Mesentery, chylous cysts of, 547; affections of, 546.
Metallic echo, 683; tinkling, 309, 683.
Metallotherapy, 1115.
Metastatic abscesses, 164.
Metasyphilitic affections, 242.
Metatarsalgia, 1106.
Meteorism in typhoid fever, 25; treatment of, 47.
Micrococci, in dengue, 99; in Malta fever, 219; in vaccine virus, 70.
Micrococcus lanceolatus, 108, 110, 644, 702.
Micrococcus melitensis, 219.
Microcytes, 798.
Micromegaly, 1145.
Micromelia, 841.
Middle cerebral artery, embolism and thrombosis of, 1011.
Migraine, 1102; treatment of, 1103.
Miliary abscesses in typhoid fever, 11.
Miliary aneurism, 998.
Miliary fever, 346; epidemics of, 347.
Miliary tubercle, 270; tuberculosis, acute, 273; tuberculosis, chronic, 295.
Milk and scarlet fever, 76; and typhoid fever, 6; products, poisoning by, 393; sickness, 344; tuberculous infection by, 267.
Mind-blindness, 990.
Mind-deafness, 990.
Miner's, anæmia or cachexia, 360; lung, 652; nystagmus, 1047; cancer of lung, 664.
Mitchell, Weir, treatment in hysteria, 1121.
Mitral incompetency, 717.
Mitral stenosis, 721; chorea and, 721; paralysis of recurrent laryngeal in, 725; presystolic murmur in, 723; rheumatism and, 721.
Moist sounds, 308.
Molluscum contagiosum, parasites in, 350.
Monophobia, 1124.
Monoplegia, cerebral, 916, 980; facial, 1052; in hysteria, 1114; in traumatic neuroses, 1134.
Montaigne on renal colic, 893.
Montreal General Hospital, autopsies in diphtheria, 143; in typhoid fever, 8; statistics, of

apex lesions in 1,000 autopsies, 332; of dysentery, 193; of hæmorrhagic small-pox, 62; of pneumonia, 131; of rheumatic fever, 167; of typhoid fever, 3.
Montreal small-pox epidemic 1885–'86, 65, 73.
Morbilli hæmorrhagici, 87.
Morbus, cæruleus, 768.
Morbus, coxæ senilis, 401, 403; errorum, 377; maculosus, 814.
Morbus maculosus neonatorum, 818.
Morphia habit, 384; treatment of, 385.
Morphinism, 384.
Morphinomania, 384.
Morphœa, 1145.
Mortality, in cerebro-spinal meningitis, 107; in pneumonia, 131; in typhoid fever, 40; in whooping-cough, 94; in yellow fever, 188.
Morton's painful foot, 1106.
Morvan's disease, 975.
Mosquitoes, relation of, to filaria disease, 361.
Motor tract, diseases of, 928.
Mountain, anæmia, 360; fever, 346; sickness, 346.
Mouth-breathing, 454.
Mouth, diseases of, 441; dry, 447; putrid sore, 442.
Movable kidney, 542, 846; dilatation of stomach in, 848.
Movable liver, 542, 587.
Mucous colitis, 544.
Mucous patches, 241.
Muguet, 443.
Multiple gangrene, 1138.
Multiple sclerosis, 959.
Mumps, 90, 447.
Munich, reduction of typhoid mortality in, 41.
Murmur, in aneurism, 780; brain, 437; cardio-respiratory, 308; in chlorosis, 794; in congenital heart-disease, 769; *Flint's*, 713; in endocarditis, 703; in lung cavity, 309; in subclavian artery in phthisis, 308; in valvular disease, 713, 716, 720, 723, 726, 727.
Musca domestica, 378; M. vomitoria, 378.
Muscle callus in sterno-mastoid in infants, 1064.
Muscle, diseases of, 1148; degeneration of, in typhoid fever, 12, 33.
Muscular atrophy, forms of, 934; heredity in, 933; atrophic and hypertrophic varieties, 935; infantile form, 935; juvenile type, 935; progressive neural form, 933; peroneal type, 933.
Muscular atrophy, progressive central, 928, 941; hereditary influence in, 929.
Muscular contractures in hysteria, 1114.
Muscular dystrophies, 933.
Muscular exertion, coma after, 869.
Muscular exertion in heart-disease, 710, 745.
Muscular rheumatism, 406.
Musculo-spiral paralysis, 1070.
Musical faculty, loss of, in aphasia, 991.
Musical murmurs, 716, 769.
Mussel poisoning, 393.
Myalgia, 406.
Myasthenia gravis pseudo-paralytica, 947.
Mycosis intestinalis, 226; pulmonum, 226.
Mycotic gastritis, 466.
Myelæmia, 802.
Myelitis, acute, 976; acute central, 977; acute transverse, 978; compression, 970; in measles, 88; reflexes in, 978; transverse, of cervical region, 979; syphilitic, 245, 246.
Myelocytes, 806.
Myelogenous leukæmia, 802.
Myiasis, 378; of nostrils and of ears, 378; of vagina, 378; cutaneous, 378; interna, 378.
Myocarditis, 748; acute interstitial, 748; fibrous, 747; in rheumatism, 171; segmenting, 21, 748; in typhoid fever, 21.
Myocardium, diseases of, 746; lesions of, due to disease of coronary arteries, 746.
Myoclonia, 1150.
Myoclonics, 1150.
Myoidema, 308.
Myopathies, the primary, 933.
Myositis, 1148; ossificans progressiva, 1149.
Myotonia, 1149; congenita, 1149.
Myotonic reaction of *Erb*, 1149.
Myriachit, 1089.
Mytilotoxin, 393.
Mytilotoxismus, 393.
Myxœdema, 840; acute, 842: congenital form, 840; operative, 842.

Nails, in typhoid fever, 18; in phthisis, 313.
Nasal diphtheria, 147.
Naso-pharyngeal obstruction, 454.
Neapolitan fever, 219.
Neck, cellulitis of, 450.
Necrosis, acute, of bone, 173; in typhoid fever, 32.
Necrosis in tubercle, 271.
Nematodes, diseases caused by, 352.
Nephralgia, 1106.
Nephritis, 869; acute, 869; after diphtheria, 150; chronic, 874; chronic hæmorrhagic, 875.
Nephritis, chronic interstitial, 877; hæmorrhages in, 882; increased tension in, 880; malarial, 209; relation of heart hypertrophy to, 879; syphilitic, 250; urine in, 880; vomiting in, 881.
Nephritis, chronic parenchymatous, 875; consecutive, 886; in erysipelas, 159; in malaria, 209; in scarlet fever, 80; in typhoid fever, 31.
Nephritis, lymphomatous, 32; suppurative, 887.
Nephrolithiasis, 891.
Nephro-phthisis (see KIDNEY, TUBERCULOSIS OF).
Nephroptosis, 542, 846.
Nephrorrhaphy, 849.

Nephrotomy, 889.
Nephro-typhus, 31.
Nerve-fibres, inflammation of, 1031.
Nerve-root symptoms, 970.
"Nerve-storms," 1103.
Nerves, diseases of peripheral, 1031; diseases of cerebral, 1038; diseases of spinal, 1067.
Nerves, lesions of anterior crural, 1072; circumflex, 1070; external popliteal, 1072; gluteal, 1072; internal popliteal, 1072; long thoracic, 1070; median, 1071; musculo-spiral, 1070; obturator, 1072; sciatic, 1072; small sciatic, 1072; ulnar, 1071.
Nervous diarrhœa, 506, 1117.
Nervous dyspepsia, 497.
Nervous system, diseases of, 901; diffuse, 951.
Nettle rash (see URTICARIA).
Neuralgia, 1104; causes of, 1104; cervico-brachial, 1105; cervico-occipital, 1067, 1105; influence of malaria in, 1104; intercostal, 1105; lumbar, 1106; of nerves of feet, 1106; phrenic, 1105; plantar, 1106; red, 1139; reflex irritation in, 1104; treatment of, 1107; trifacial, 1105; visceral, 1106.
Neurasthenia, 1122; sexual, 1126; traumatic, 1132.
Neuritis, 1031; arsenical, 1035; fascians, 1032; interstitial, 1031; of infants, progressive interstitial hypertrophic, 951; lipomatous, 1031; localized, 1031, 1032; parenchymatous, 1031; multiple, 1031, 1033; alcoholic, 1034; endemic, 220, 1031; in diphtheria, 151; in chronic phthisis, 312; in the infectious diseases, 1034; in typhoid fever, 29; recurring, 1033; saturnine, 1035; traumatic, 1032; optic, 1040.
Neuroglioma, 1020.
Neuroma, plexiform, 1037.
Neuromata, 1037.
Neuroses, occupation, 1107; traumatic, 1132.
Neutrophiles, 806.
New-born, hæmorrhagic diseases of, 818.
New growths in the bowel, 533.
Night-blindness, 1040; in scurvy, 824.
Night-sweats in phthisis, 306; treatment of, 337.
Night-terrors, 455.
Nipple, *Paget's* disease of, 350.
Nits, 377.
Nodding spasm, 1066.
Nodes, *Heberden's*, 401.
Nodes, symmetrical, in congenital syphilis, 244.
Nodules, rheumatic, 172.
Noma, 444; in scarlet fever, 82; in typhoid fever, 33, 35.
Normoblasts, 794, 799.
Nose, bleeding from (see EPISTAXIS), 614.
Nose, diseases of, 610.
Nummular sputa in phthisis, 300.
Nurse's contracture of *Trousseau*, 1110.
Nutmeg liver, 553.
Nyctalopia, 1040; in scurvy, 824.
Nystagmus, 1047; in *Friedreich's* ataxia, 950; in insular sclerosis, 959; of miners, 1047.

Obesity, 439.
Obsession, 1089.
Obstruction of bowels, 531; acute, 534; chronic, 535.
Obturator nerve, affections of, 1072.
Occipital lobes, tumors of, 1022.
Occipito-cervical neuralgia, 1067, 1105.
Occupation neuroses, 1107.
Ocular palsies, treatment of, 1050.
Oculo-motor paralysis, recurring, 1046.
Odor, in small-pox, 68; in typhoid fever, 18.
Œdema, angio-neurotic, 1140; febrile purpuric, 816; hereditary, 1141; of lungs, 636; of brain, 997; in uræmia, 866, 997.
Œdematous laryngitis, 617.
Oertel's method in obesity, 439, 752.
Œsophageal *bruit*, 461.
Œsophago-pleuro-cutaneous fistula, 462.
Œsophagismus, 459.
Œsophagitis, acute, 458; chronic, 459.
Œsophagus, diseases of, 458; cancer of, 461; dilatations of, 462; diverticula of, 462; hæmorrhage from in cirrhosis of liver, 572; paralysis of, 459; post-mortem digestion of, 462; rupture of, 462; spasm of, 459; stricture of, 460; syphilis of, 249; tuberculosis of, 318; ulceration of, 459; varices of veins, in cirrhosis of liver, 572.
Oïdium albicans, 443.
Olfactory nerves and tracts, diseases of, 1038.
Omentum, tuberculous tumor of, 287; tumor of, in cancer of the peritonæum, 605.
Omodynia, 407.
Onomatomania, 1089.
Onychia, in arthritis deformans, 402; in locomotor ataxia, 925; syphilitic, 241, 243.
Operation *per se*, effects of, in epilepsy, 1101.
Operation, tuberculosis after, 270.
Ophthalmia, gonorrhœal, with arthritis, 173.
Ophthalmoplegia, 942, 1049; externa, 1049; interna, 1049.
Opisthotonos, cervical, in infants, 957; in tetanus, 232.
Opium, poisoning, diagnosis from uræmia, 868; habit, 384; smoking, effects of, 384.
Optic nerve atrophy, 1041; hereditary, 1041; primary, 1041; secondary, 1041; in tabes, 922.
Optic nerve and tract, diseases of, 1039.
Optic neuritis, 1040; in abscess of brain, 1026; in brain-tumor, 1021; in tuberculous meningitis, 278.
Orchitis, in malaria, 217; in mumps, 91; interstitial, in syphilis, 251; in typhoid fever, 32;

in variola, 58; parotidea, 91; tuberculous, 326; value of, in diagnosis, 326.
Orthotonos, in tetanus, 232.
Osteitis deformans, 1144.
Osteo-arthropathy, hypertrophic pulmonary, 1144.
Osteo-myelitis simulating acute rheumatism, 173.
Otitis-media, in typhoid fever, 30; in scarlet fever, 81; in meningitis, 106.
Ovaries, tuberculosis of, 326.
Over-exertion, heart affections due to, 745.
Oxalate-of-lime calculus, 892.
Oxaluria, 861.
Oxygen, inhalations of, in diabetic coma, 431; in pneumonia, 137.
Oxyuris vermicularis, 353.
Oysters and typhoid fever, 6.
Oysters, poisoning by, 393; and typhoid fever, 6.
Ozæna, 612.

Pachymeningitis, 951.
Pachymeningitis cervicalis hypertrophica, 953.
Pachymeningitis hæmorrhagica, of cerebral dura, 952; of spinal dura, 953.
Palate, paralysis of, in diphtheria, 150; in facial paralysis, 1053; perforation of, in scarlet fever, 82.
Palate, tuberculosis of, 318.
Palpable kidney, 846.
Palpitation of heart, 755.
Palsies, cerebral, of children, 938, 1017.
Palsy, lead, 388.
Paludism (see Malarial Fever), 202.
Pancreas, cancer of, 594; in diabetes, 421; cysts of, 592; hæmorrhage into, 588; tumors of, 594.
Pancreas, diseases of, 588.
Pancreatic abscess, 590; diabetes, 422; calculi, 595.
Pancreatitis, acute hæmorrhagic, 589; chronic, 592; fat necrosis in, 591; gangrenous, 590; suppurative, 590.
Pantophobia, 1124.
Papillitis, 1040.
Paræsthesia (numbness and tingling), in neuritis, 1032; in locomotor ataxia, 924; in tumor of brain, 1022; in primary combined sclerosis, 949.
Parageusis, 1060.
Paralysis, acute ascending, 946; acute spinal, of adults, 946; acute, of infants, 942; agitans, 1076; alcoholic, 1034; anæsthesia, 1035; asthenic bulbar, 947; atrophic spinal, 942; *Bell's*, 1051; bulbar, acute, 933; chronic, 933; of bladder, in myelitis, 977; of brachial plexus, 1069; in chorea, 1083; of circumflex nerve, 1070; crossed or alternate, 984, 1004; "crutch," 1070; *Cruveilhier's*, 929; diver's, 969; of diaphragm, 1068; after diphtheria, 150; following epilepsy, 1097; of facial nerve, 1051; of fifth nerve, 1050; of fourth nerve, 1047; general, of the insane, 960; of hypoglossal nerve, 1066; hysterical, 1113; infantile, 942; labio-glosso-laryngeal, 932; *Landry's*, 946; of laryngeal abductors, 1061; of adductors, 1061; in lateral sclerosis, 937; from lead, 388; in locomotor ataxia, 925; of long thoracic nerve, 1070; in meningitis, 278, 956; of median nerve, 1071; of musculo-spiral nerve, 1070; of oculomotor nerves, 1046; of olfactory nerve, 1038; periodical, 1136; in progressive muscular atrophy, 930; radial, 1070; of rectum, in myelitis, 977; of recurrent laryngeal nerve, 1061; secondary to visceral disease, 1032; of sixth nerve, 1048; of third nerve, 1046; of ulnar nerve, 1071; of vocal cords, 1061.
Paramyoclonus multiplex, 1150.
Paraphasia, 992.
Paraplégia flasque, 941.
Paraplegia, from alcohol, 1034; ataxic, 948; from anæmia of spinal cord, 966; from compression of cord, 970; from hæmorrhage into cord, 968; hereditary form of, 940; hysterical, 941, 1114; in lathyrism, 394; from myelitis, 977; in pellagra, 395; spastic, 937; spastica cerebralis, 938; syphilitic, 940; from tumor of the cord, 974; in tabes, 925.
Parasites, diseases due to animal, 349.
Parasitic gastritis, 466.
Parasitic stomatitis, 443.
Parasyphilitic affections, 242, 961.
"Parchment crackling" in rickets, 435.
Parenchymatous nephritis, 875.
Parieto-occipital region, brain tumors in, 1022.
"Paris green," poisoning by, 390.
Parkinson's disease, 1076.
Parosmia, 1038.
Parotid bubo, 447.
Parotitis, epidemic, 90; deafness in, 91; delirium in, 91; chronic, 447; orchitis in, 91; specific, 447.
Parotitis, symptomatic, 447; after abdominal section, 447; in pneumonia, 125; in typhoid fever, 23; in typhus fever, 52.
Paroxysmal hæmoglobinuria, 853.
Parrot's ulcers, 443.
Parry's disease, 836.
Patellar-tendon reflex (see Knee-jerk).
Pathophobia, 1124.
Pectoriloquy, 309.
Pediculi, 376; relations of, to tache bleuâtre, 18, 377.
Pediculosis, 376.
Pediculus capitis, 376; P. corporis, 377.
Peliomata, 17.
Peliosis rheumatica, 815; in chorea, 1085.
Pellagra, 395.
Pelvis of kidney, affections of (see Pyelitis).

Tuberculosis

Pemphigoid purpura, 815.
Pemphigus neonatorum, 242.
Pentastomes, 375.
Peptic ulcer, 478; dyspepsia in, 481; hæmorrhage in, 481; pain in, 481; tenderness on pressure in, 482.
Peptones in the urine, 857.
Perforating ulcer of foot in tabes, 925; in diabetes, 425.
Perforation of bowel in dysentery, 200; in typhoid fever, 10, 25.
Periarteritis, gummatous, 250; nodosa, 788.
Pericardial friction, 690.
Pericarditis, 688; acute plastic, 689; aphonia in, 692; chronic adhesive, 696; delirium in, 692; dysphagia in, 692; epidemics of, 689; epilepsy in, 693; from extension of disease, 689; from foreign body, 688; in chorea, 1084; in fœtus, 689; in gout, 415; in rheumatism, 171; hæmorrhagic, 692; hyperpyrexia in, 690, 692; mental symptoms in, 692; primary, 688; pulsus paradoxus in, 692; secondary, 688; with effusion, 691.
Pericardium, adherent, 696; *Friedreich's* sign in, 697.
Pericardium, diseases of, 688; tuberculosis of, 285; air in, 698.
Perichondritis, laryngeal, in typhoid fever, 27; in tuberculosis, 619.
Perihepatitis, 575, 603.
Perinephric abscess, 900.
Perinuclear basophilic granules, 410.
Periodical paralysis, 1136.
Periosteal cachexia, 825.
Peripheral neuritis, 1031.
Peristaltic unrest, 498, 1117.
Peritonæum, diseases of, 596.
Peritonæum, fluid in, 605, 609; cancer of, 604; new growths in, 604.
Peritonæum, tuberculosis of, 286.
Peritonæum, tumor formations in tuberculosis of, 287.
Peritonitis, acute general, 522, 526, 596; appendicular, 526, 602; chronic, 602; chronic hæmorrhagic, 604; diffuse adhesive, 603; hysterical, 599; idiopathic, 596; in infants, 600; in typhoid fever, 26; leukæmic, 805; local adhesive, 602; localized, 522, 600; pelvic, 602; perforative, 596; primary, 596; proliferative, 603; pyæmic, 596; rheumatic, 596; secondary, 596; septic, 596; subphrenic, 600; tuberculous, 286, 604.
Peritonitis, tuberculous, effects of operation on, 609.
Perityphlitis, 519.
"Perles" of *Laennec*, 630.
Pernicious anæmia, 795.
Pernicious malaria, 207, 215.
Peroneal type of muscular atrophy, 933.
Pertussis (see Whooping-cough), 92.
Pesta magna, 56.
Pestis minor, 191; major, 191; siderans, 191.
Petechiæ in epilepsy, 1097; in relapsing fever, 54; in scurvy, 823; in small-pox, 62; in typhus fever, 51.
Petechial fever, 101.
Petit mal, 1094, 1097; in general paresis, 962.
Peyer's patches in typhoid fever, 8; in measles, 86; in tuberculosis, 319.
Phagocytosis in erysipelas, 158; in malaria, 207; in tuberculosis, 271.
Pharyngitis, 448; acute, 448; chronic, 449; sicca, 449.
Pharynx, acute infectious phlegmon of, 450; hæmorrhage into, 448; hyperæmia of, 448; œdema of, 448; paralysis of, 1060; spasm of, 1061; tuberculosis of, 318; ulceration of, 449.
Pharynx, diseases of, 448.
Philadelphia Hospital, relapsing fever at, in 1844, 53; typhoid and typhus fever at, 2; typhus epidemic in 1883, 49; *statistics* of cerebro-spinal fever, 104; of delirium tremens in, 383.
Philadelphia Infirmary for Nervous Diseases, *statistics* of chorea, 1079; of epilepsy, 1094.
Philadelphia, tuberculosis in city wards, 266; yellow-fever epidemic in 1793, 182.
Phlebitis of portal vein, 577.
Phlebo-sclerosis, 773.
Phosphates, alkaline, 862; earthy, 862.
Phosphatic calculi, 892.
Phosphaturia, 862.
Phosphorus poisoning, similarity of acute yellow atrophy to, 553.
Phrenic nerve, affections of, 1068.
Phthiriasis, 376.
Phthirius pubis, 377.
Phthisical frame, *Hippocrates'* description of, 268.
Phthisis, 289; chronic ulcerative, 294; acute pneumonic, 289; arterio-sclerosis in, 316; basic form of, 295; Bright's disease in, 312; of coal-miners, 269, 652; chronic arthritis in, 316; cough in, 300; endocarditis in, 298, 310; diagnosis of, 313; distribution of lesions in, 294; erysipelas in, 315; fatal hæmorrhage in, 317; fever in, 304; forms of cavities in, 296; gastric symptoms of, 311; hæmoptysis in, 302; modes of death in, 317; modes of onset in, 298; physical signs of, 306; pneumonia in, 315; relation of fistula in ano to, 320; sputum in, 300; summary of lesions in, 295; typhoid fever in, 315; vomiting in, 311.
Phthisis, fibroid, 314, 649; florida, 292; renum, 324; syphilitic, 247; of stone-cutters, 269, 652; unity of, 272; ventriculi, 467.
Physiological albuminuria, 855.
Pia mater, diseases of, 954.
Picric-acid test for albumin, 857.

Pigeon-breast, in rickets, 436; in mouth-breathers, 455.
Pigmentation of skin, from arsenic, 390; in *Basedow's* disease, 839; from phthiriasis, 377; in *Addison's* disease, 830; in chronic pulmonary tuberculosis, 313; in melanosis, 831; in peritoneal tuberculosis, 287; in scleroderma, 1146.
Pigmentation of viscera in pellagra, 395.
Pigs, tuberculosis in, 258.
Pin-worms, 353.
Pitting in small-pox, 61; measures to prevent, 67.
Pituitary body in acromegaly, 1143; in gigantism, 1143.
Pityriasis versicolor, 313.
Plague, bubonic, 189.
Plague spots, 192.
Plaques jaunes, 1009.
Plastic bronchitis, 633.
Pleura, diseases of, 665.
Pleura, echinococcus of, 373; tuberculosis of, 284.
Pleural effusion, *Baccelli's* sign in, 670, 672; compression of lung in, 667; hæmorrhagic, 673; in scarlet fever, 81; position of heart in, 668; pseudo-cavernous signs in, 670; purulent, 671; serous effusion, constituents of, 667; sudden death in, 671.
Pleural membranes, calcification of, 679.
Pleurisy, acute, 665; diaphragmatic, 674; encysted, 674; fibrinous, 665; interlobar, 674; in typhoid fever, 28; pain in side in, 668; plastic, 665; pleural friction in, 670; pulsating, 672; purulent, 671; sero-fibrinous, 666; tuberculous, 284, 666, 673.
Pleurisy, chronic, 678; dry, 679; primitive dry, 679; vaso-motor phenomena in, 680; with effusion, 678.
Pleurodynia, 407.
Pleuro-peritoneal tuberculosis, 284.
Pleurothotonos in tetanus, 232.
Plexiform neuroma, 1037.
Plica polonica, 377.
Plumbism, 386; and gout, 408; as a cause of renal cirrhosis, 877; paralysis in, 388.
Plymouth, epidemic of typhoid fever at, 5.
Pneumatosis, 499.
Pneumaturia, 424, 864.
Pneumogastric auræ, 1096.
Pneumogastric nerve, affections of, 1060; cardiac branches of, 1062; gastric and œsophageal branches of, 1063; laryngeal branches of, 1061; pharyngeal branches of, 1060; pulmonary branches of, 1063.
Pneumonia, acute croupous, 108; abscess in, 130; acute delirium in, 123; antipneumonic serum in, 135; bleeding in, 135; clinical varieties of, 126; colitis, croupous, in, 115; complications of, 123; crisis in, 117; delayed resolution in, 129; diagnosis from acute pneumonic phthisis, 291; diplococcus pneumoniæ, 110; endocarditis in, 115; engorgement of lung in, 113; epidemics of, 112, 127; fever of, 116; gangrene in, 130; gray hepatization in, 113; herpes in, 122; immunity from, 112; in diabetes, 127; in infants, 126; in influenza, 128; in old age, 126; meningitis in, 115; mortality of, 131; pericarditis in, 115; pseudo-crisis in, 117; purulent infiltration in, 113; recurrence of, 125; red hepatization in, 113; relapse in, 125; resolution of, 113; toxæmia in, 132; trauma in, 109.
Pneumonia, acute syphilitic, 248; apex pneumonia, 126; aspiration or deglutition, 642; central, 126; "cerebral," 122; chronic interstitial, 649; chronic pleurogenous, 680; contusion, 109; double, 126; ether, 129; epidemic, 127; fibrinous, 108; hypostatic, 635; in malaria, 209; interstitial, of the root, in syphilis, 247; in typhoid fever, 27; larval, 127; lobar, 108; massive, 126; migratory, 126; pleurogenous interstitial, 649; post-operation, 128; secondary, 127; typhoid pneumonia, 127; white, of the fœtus, 247.
Pneumonitis, 108.
Pneumonokoniosis, 652.
Pneumo-pericardium, 698.
Pneumo-peritonæum, 598.
Pneumorrhagia, 637.
Pneumothorax, 681; after tracheotomy, 687; chronic, 683; Hippocratic succussion in, 683; in phthisis, 297; from muscular effort, 681.
Pneumotoxin, 112.
Pneumo-typhus, 11, 28.
Podagra, 407.
Pododynia, 1106.
Poikilocytosis, 794, 799.
Poisoning, by food, 391; by lead, 386; by meat, 391; by sewer-gas, 343.
Polio-myelitis, acute and subacute, in adults, 946.
Polio-myelitis anterior, acute, 942; epidemics of, 942; etiology of, 942; morbid anatomy of, 943; prognosis of, 944; symptoms of, 943.
Polio-myelitis anterior chronica, 928, 941.
Polyadenomata, 494.
Polyæmia, 803.
Polyneuritis, acute febrile, 1033; recurrens, 1033.
Polyphagia, 423.
Polysarcia, 439.
Polyuria (see Diabetes Insipidus), 432.
Polyuria, in abdominal tumors, 432; in hysteria, 432, 1112.
Pons, lesions of, 984; tumors of, 1023.
Popliteal nerve, external, 1072; internal, 1072.
Porencephalus, 1017.
Portal vein, 554; thrombosis of, 554; suppuration in, 578.

Post-epileptic symptoms, 1097.
Post-hemiplegic chorea, 1019; epilepsy, 1019, 1098; movements, 1019.
Post-mortem movements in cholera bodies, 178.
Post-pharyngeal abscess, 450.
Post-typhoid, anæmia, 19; elevations of temperature, 16.
Pott's disease, 970.
Pregnancy, and acute yellow atrophy, 551; and chorea, 1080; and heart-disease, 729; and phthisis, 329; and typhoid fever, 35.
Presystolic murmur, 723.
Priapism in leukæmia, 805.
Prickly heat (see Urticaria).
Procession caterpillar, effects of, 379.
Professional spasms, 1107.
Proglottis of tænia, 365.
Progressive muscular atrophy, 928.
Progressive pernicious anæmia, 795; blood in, 797.
Prophylaxis against cholera, 180; against scurvy, 824; against tuberculosis, 330; against tænia, 367; against trichina, 359; against typhoid fever, 41; against yellow fever, 188.
Prosopalgia, 1105.
Prostate, tuberculosis of, 326.
Protozoa, diseases caused by, 349; parasitic, 349.
Prune-juice expectoration, 664.
Prurigo, in *Hodgkin's* disease, 812.
Pruritus in diabetes, 425; in uræmia, 867; in obstructive jaundice, 549; in gout, 415; in *Graves'* disease, 839.
Pseudo-angina pectoris, 763, 1118.
Pseudo-apoplectic seizures in fatty heart, 751; with slow pulse, 760.
Pseudo-biliary colic, 564.
Pseudo-bulbar paralysis, 932.
Pseudo-cavernous signs, 309, 670, 675.
Pseudo-cyesis, 1114.
Pseudo-diphtheria, 142.
Pseudo-hydrophobia, 230.
Pseudo-leukæmia, 809.
Pseudo-lipoma, supraclavicular, 841.
Pseudo-ptosis, 1046.
Pseudo-sclérose en plaques, 960.
Pseudo-tuberculosis hominis streptothrica, 262.
Psilosis, 511.
Psoriasis, buccal, 446.
Psorospermiasis, 349.
Ptosis, forms of, 1046; hysterical, 1046; in ataxia, 922; pseudo-, 1046.
Ptyalism, 444, 446.
Puberty, barking cough of, 1117.
Pulex, irritans, 377; penetrans, 378.
Pulmonal-cerebral abscesses, 1025.
Pulmonary (see Lungs).
Pulmonary apoplexy, 638.
Pulmonary artery, sclerosis of, 773; perforation of, 782.
Pulmonary hæmorrhage, 637.
Pulmonary orifice, congenital lesions of, 767; tuberculosis in, 316, 767; valve, lesions of, 727.
Pulmonary osteo-arthropathy, hypertrophic, 1144.
Pulsating pleurisy, 672.
Pulsation, dynamic, of aorta, 782.
Pulse, alternate, 757; anastomotic, 774; dicrotic, 13, 20; under influence of digitalis, 732; intermittent, 757; irregular, 757; bigeminal, 757; recurrent, 774; trigeminal, 757.
Pulse, capillary (see Capillary); *Corrigan*, 714; water-hammer, 714.
Pulse, slow, in tuberculous meningitis, 279; in jaundice, 549 (see Brachycardia, 759).
Pulsus paradoxus, 692, 697, 756.
Pupil, *Argyll Robertson*, 962, 1047.
Pupillary inaction, hemiopic, 1044.
Pupils, unequal, 1047; in general paresis, 962.
Purpura, 814; arthritic, 815; cachectic, 814; fulminans, 817; *Henoch's*, 816; infectious, 814; mechanical, 815; neurotic, 815; peliosis rheumatica in, 815; hæmorrhagica, 816; pemphigoid, 815; simplex, 815; symptomatic, 814; toxic, 814; urticans, 815; variolosa, 62.
Purpuric œdema, 816.
Pustule, malignant, 225.
Putrid sore mouth, 442.
Pyæmia, 163; arterial, 705; idiopathic, 163; post-typhoid, 33.
Pyæmic abscess of liver, 579, 580.
Pyelitis, 886; intermittent fever in, 888; pyuria in, 887; in typhoid fever, 32.
Pyelonephritis, 886.
Pylephlebitis adhesiva, 554.
Pylephlebitis, in dysentery, 200; in pyæmia, 164; suppurative, 555, 578.
Pylorus, hypertrophic stenosis of, 494; congenital hypertrophy of, 494; insufficiency of, 500; spasm of, 499.
Pyonephrosis, 886.
Pyo-pneumothorax, 285, 681.
Pyo-pneumothorax subphrenicus, 479, 601, 683.
Pyuria, 858; in typhoid fever, 11, 32.

Quarantine against yellow fever, 188; against cholera, 180.
Quartan ague, 213.
Quincke's lumbar puncture, 107, 956.
Quinine rash, 77, 83.
Quinsy (see Tonsillitis, Suppurative).
Quotidian ague, 213.

Rabies, 227.
Rachitic bones, 434.
Rachitis (see Rickets), 434.
Radial paralysis, 1070.
Rag-picker's disease, 226.
Railway brain, 1132.

Railway spine, 1132.
Rainey's tubes, 349.
Rapid heart, 758.
Rashes, from drugs, 83, 814; in glanders, 234; in measles, 86; in relapsing fever, 54; in rubella, 89; in scarlet fever, 77; in small-pox, 59, 60; in syphilis, 240; in typhoid fever, 17; in typhus fever, 51; in pyæmia, 164; in vaccination, 71; in varicella, 74.
Raspberry tongue in scarlet fever, 78.
Ray-fungus (actinomyces), 235.
Raynaud's disease, 1137; aphasia in, 1139; and scleroderma, 1146; epilepsy in, 1139; hæmoglobinuria in, 1138.
Reaction of degeneration, 914, 1036, 1054
Recrudescence of fever in typhoid fever, 16.
Rectal crises in tabes, 924.
Rectum, irritable, 1118; stricture of, 249; syphilis of, 249; tuberculosis of, 320.
Recurrent laryngeal nerve, paralysis of, 1061.
Recurrent pulse, 774.
Recurring multiple neuritis, 1033.
Red softening of brain, 1009.
Reduplication of heart-sounds, 757.
Redux crepitus, 120.
Reflex epilepsy, 1095.
Reflexes in ascending paralysis, 946; in cerebral hæmorrhage, 1005, 1006; in locomotor ataxia, 924; in polio-myelitis acuta, 944; in spastic paraplegia, 937; in hysterical paraplegia, 941, 1114; in progressive muscular atrophy, 931.
Regurgitation, tricuspid, 725.
Reichmann's disease, 500.
Relapse in typhoid fever, 36.
Relapsing fever, 53; spirillum of, 54.
Remittent fever, 213.
Renal calculus, 891.
Renal, colic, 893; epistaxis, 852; sand, 892; syphilis, 250; sclerosis, 877.
Rendu's type of tremor, 1115.
Ren mobilis, 846.
Resolution in pneumonia, 129.
Resonance, amphoric, 309, 682; tympanitic, 309, 669, 682.
Respiratory system, diseases of, 610.
Rest treatment, 1121; in aneurism, 784.
Retina, lesions of, 1039.
Retinal hyperæsthesia, 1040.
Retinitis, albuminuric, 1039; in anæmia, 1039; in malaria, 1039; leukæmic, 1040; pigmentosa, 1039; syphilitic, 241, 1039.
Retraction of head in meningitis, 278, 955.
Retro-collic spasm, 1065.
Retroperitoneal abscess, 522.
Retroperitonæum, hæmorrhage into, 58.
Retro-pharyngeal abscess, 450.
Retropulsion in paralysis agitans, 1078.
Revaccination, 71.
Rhabditis Niellyi, 361.
Rhabdo-myoma of kidney, 896.
Rhabdonema intestinale, 364.
Rhagades, 242.
Rheumatic fever, 166; cerebral complications of, 171; endocarditis in, 170; fibrous nodules in, 172; germ theory of, 168; heredity in, 167; hyperpyrexia in, 170; metabolic theory of, 168; nervous theory of, 168; pericarditis in, 171; purpura in, 172; sudden death in, 172.
Rheumatic gout (see Arthritis Deformans).
Rheumatic nodules, 172.
Rheumatism, chronic, 405.
Rheumatism, muscular, 406.
Rheumatism, subacute, 170.
Rheumatoid arthritis (see Arthritis Deformans).
Rhinitis, 611; atrophica, 611; fibrinosa, 147; hypertrophica, 611; syphilitic, 242.
Ribs, resection of, in empyema, 678.
Rice-water stools, 179.
Rickets, 434; acute, 438, 825; fœtal, 841.
Riga's disease, 442.
Rigidity, early, in hemiplegia, 1002.
Rigidity, late, in hemiplegia, 1005.
Rigors, in abscess of brain, 1026; in abscess of liver, 579; in ague, 209; in pneumonia, 115; in pyæmia, 164; in pyelitis, 887; in tuberculosis, 299; in typhoid fever, 17.
Risus sardonicus, 232.
Rock-fever, 219.
Romberg's symptom, 923.
Root-nerve symptoms in compression paraplegia, 970.
Rosary, rickety, 436.
Roseola (see Rose Rash of Typhoid), 17; epidemic, 89.
"Rose cold," 612.
Rose rash in typhoid fever, 17.
Rotation in epilepsy, 1096.
Rotatory spasm in hysteria, 1115.
Rötheln, 89.
"Rough-on-rats," poisoning by, 890.
Round-worms, 352.
Rub (see Friction).
Rubella, 89.
Rubeola notha, 89.
Rumination, 499.
Running pulse in typhoid fever, 20.
Russian fever, 95.

Sable intestinal, 546.
Saccharomyces albicans, 443.
Sacral plexus, lesions of, 1072.
St. Vitus's dance, 1079.
Salaam convulsions, 1091, 1115.
Saline injections, intravenous, in diabetic coma, 431; subcutaneous, in cholera, 181.
Saliva, arrest of, 447; supersecretion of, 446.

Salivary glands, diseases of, 446; inflammation of, 447.
Salivation (see Ptyalism), 444, 446; in small-pox, 61; in bulbar paralysis, 932.
Salpingitis, tuberculous, 326.
Saltatory spasm, 1089.
Sanatoria, treatment of tuberculosis in, 333.
Sand-flea, 378.
Sapræmia, 161.
Saranac Sanitarium, 333.
Sarcina, ventriculi, 475; in lung cavities, 302.
Sarcocystis Miescheri, 349; sarcocystis hominis, 349.
Sarcoma, of brain, 1020; of kidney, 896; of liver, 583; of lung, 663; mediastinal, 685; melanotic, of liver, 583.
Sarcoptes scabiei, 376.
Saturnine neuritis, 1035.
Saturnism, 386.
Sausage poisoning, 391.
Scapulodynia, 407.
Scarlatina, miliaris, 78.
Scarlatina sine eruptione, 79.
Scarlatinal nephritis, 80.
Scarlet fever, 75; anginose form, 80; atactic form, 79; complications and sequelæ, 80; contagiousness of, 76; desquamation in, 79; eruption in, 77; hæmorrhagic form, 79; incubation of, 77; invasion in, 77; malignant, 79; puerperal, 76; surgical, 76.
Schistosoma hæmatobium, 352.
Schönlein's disease, 815.
School-made chorea, 1081.
Schott treatment in myocardial disease, 752.
Sciatica, 1073.
Sciatic nerve, affections of, 1072.
Scirrhous cancer of stomach, 487, 488.
Sclerema in cholera infantum, 510.
Sclerema neonatorum, 1145.
Sclerodactylie, 1146.
Scleroderma, 1145.
Sclérose en plaques, 959.
Scleroses of the brain, 957.
Sclerosis, cerebro-spinal, 957; degenerative, 957; developmental, 958; inflammatory, 958; of scurvy, 823; syphilis as a cause of, 242.
Sclerosis, primary, lateral, 937; insular, 959; multiple, 959.
Sclerosis, posterior spinal (see Locomotor Ataxia), 920; in chronic ergotism, 394.
Sclerosis, primary combined, 949.
Sclerosis in tubercles, 271.
Sclerosis, renal, 877.
Sclerosis, toxic combined, 951.
Sclerostomum duodenale, 359; S. equinum, 359.
Sclerotic gastritis, 467.
Scolices of echinococcus, 371.
Scorbutus, 821.
Scrivener's palsy, 1107.
Scrofula, 280; alleged protective inoculation by, 281.
Scrofulous pneumonia, 272.
Scurvy, 821; infantile, 825; prophylaxis of, 824; sclerosis, 823.
Scybala, 539.
Seasonal relations, of chorea, 1079; of malaria, 204; of pneumonia, 110; of rheumatism, 167.
Secondary contracture in hemiplegia, 1005.
Secondary deviation, 1048.
Secondary fever of small-pox, 60.
Self-limitation in tuberculosis, 328.
Semilunar space of *Traube*, 669.
Semilunar valves, aortic, incompetency of, 709.
Senile emphysema, 659.
Sensation, painful, loss of, in syringomyelia, 975.
Sensation, retardation of, in ataxia, 924.
Sensory system, diseases of, 920.
Septicæmia, 160; cryptogenetic, 162; general, 162; gonorrhœal, 255; progressive, 162; post-typhoid, 33.
Septico-pyæmia, 163.
Serratus palsy, 1070.
Seven-day fever, 53.
Sewer-day and tonsillitis, 451.
Sewer-gas poisoning, effects of, 343.
Sex, influence of, in heart-disease, 729.
Sexes, proportion of, affected with acute yellow atrophy, 551; in chlorosis, 792; in chorea, 1079; in exophthalmic goitre, 837; in general paresis, 960; in hæmophilia, 819.
Shaking palsy, 1076.
Shell-fish, poisoning by, 393.
Ship-fever, 49.
Shock as a cause of traumatic neuroses, 1132.
Shock, death from, in acute obstruction, 535.
Sick headache, 1102.
Sickness, sleeping, 361.
Siderodromophobia, 1124.
Siderophobia, 1124
Siderosis, 652, 654.
Signal symptom (in cortical lesions), 980, 1021.
Singultus (see Hiccough).
Sinus thrombosis, 1015; and anæmia, 1015; in chlorosis, 794; autochthonous, 1015; pyæmia, 1015; secondary, in ear-disease, 1015.
Siriasis, 395.
Sitotoxismus, 394.
Sixth nerve, paralysis of, 1048.
Skin, itching of, in uræmia, 867.
Skoda's resonance in pleural effusion, 669; in pneumonia, 119.
Skull, of congenital syphilis, 243; of hydrocephalus, 1029; of rickets, 436; percussion of, 1027.
Sleeping sickness, 361.
Slow heart, 759.
Small pox, 56; complications of, 64; confluent

form, 61; contagiousness of, 56; discrete form, 60; eruption in, 60; hæmorrhagic, 62; inoculation in, 56; vaccination in, 56.
Small sciatic nerve, affections of, 1072.
Smell, affections of sense of (see Olfactory Nerve), 1038.
Snake-virus, purpura caused by, 814.
Snuffles, 242.
Softening of brain, 1008.
Soil, influence of, in cholera, 177; in tuberculosis, 268; in typhoid fever, 6.
Solvent treatment of renal calculi, 896.
Soor, 443.
Sordes, 22.
Sore throat, 448.
Soya bread, 430.
Spasm, congenital gastric, 495.
Spasm, lock, in writer's cramp, 1108.
Spasmodic wryneck, 1065.
Spasms, in ergotism, 894; in hydrophobia, 228; in hysteria, 1112; of face, 1055; of muscles, after facial paralysis, 1055; professional, 1107; saltatory, 1089.
Spastic paraplegia of adults, 937; hereditary, 940; hysterical, 941; *Erb's* syphilitic, 940; in children, 938; secondary, 941.
Specific infectious diseases, 1.
Specific treatment of typhoid fever, 47.
Spectra, fortification, 1102.
Speech (see Aphasia), 988.
Speech, in adenoid vegetations, 456; in bulbar paralysis, 932; in insular sclerosis, 959; in general paralysis, 962; in hereditary ataxia, 950; in paralysis agitans, 1078.
Speech, scanning, in insular sclerosis, 959.
Spes phthisica, 312.
Spina bifida, involvement of cauda equina in, 972.
Spinal accessory nerve, paralysis of, 1063.
Spinal apoplexy, 968.
Spinal concussion, effects of, 1133.
Spinal cord, diffuse and focal diseases of, 964.
Spinal cord, abscess of, 974; affections of blood-vessels of, 966; anæmia of, 966; chronic leptomeningitis of, 957; compression of, 970; congestion of, 966; embolism and thrombosis of vessels of, 966; endarteritis of vessels of, 967; fissures in, 969; hæmorrhage into, 968; leptomeningitis of, 954; localization of functions of, 905; pachymeningitis of, 953; sclerosis, primary combined, of, 949; syphilis of, 244; tuberculosis of, 321; tumors of, 973; unilateral lesions of, 965.
Spinal epilepsy, 937.
Spinal irritation, 1125.
Spinal membranes, hæmorrhage into, 967.
Spinal nerves, diseases of, 1067.
Spinal neurasthenia, 1125.
Spinal paralysis, atrophic, 942.
Spirals, *Curschmann's*, 631, 634.
Spirillum of relapsing fever, 54.
Spirochæte of Obermeier, 53.
Splanchnoptosis, 541.
Spleen, amyloid degeneration of, in syphilis, 249; in tuberculosis, 298.
Spleen, diseases of, 832; abscess of, 834; infarct of, 834; tumors of, 834.
Spleen, enlargement of, in congenital syphilis, 242, 244; in malaria, 207, 216.
Spleen, excision of, in hypertrophy, 809; in leukæmia, 809.
Spleen, floating, 543, 833; excision of, 809.
Spleen, in ague, 208, 216; in anthrax, 226; in cirrhosis of liver, 572, 575; in *Hodgkin's* disease, 811; hydatid of, 372; in leukæmia, 803, 805; in rickets, 435, 437; in acute tuberculosis, 276; in typhoid fever, 10, 26; in typhus, 50.
Spleen, puncture of, 40.
Spleen, rupture of, 833; in malaria, 207; in typhoid fever, 11, 26.
Splenectomy, statistics of, 809.
Splenic anæmia, 834.
Splenic fever, 224.
Splenization of lung, 292, 643.
Spleno-megaly, primitive, 834.
Spondylitis deformans, 403.
Sporozoa, 349; parasitic, 349.
Spotted fever, 49, 101.
Sprue, 511.
Sputa, albuminoid, after aspiration of chest, 678; alveolar cells in, 622, 635; amœba coli in, 201; in cancer of lung, 664; in influenza, 97; hæmatoidin crystals in, 580; in anthracosis, 654; in asthma, 630; in bronchiectasis, 627; in acute bronchitis, 622; in chronic bronchitis, 624; in putrid bronchitis, 625; in gangrene of lung, 661.
Sputa, in phthisis, 300; in pneumonia, 118; in acute pulmonary tuberculosis, 275; prune-juice, 664; uric-acid crystals in, 411.
Staphylococci, in diphtheria, 141; in endocarditis, 702; in peritonitis, 597; in pneumonia, 113; in pyæmia, 163; in rheumatic fever, 168; in septicæmia, 162; in tonsilitis, 451.
Status, epilepticus, 1097; hystericus, 1119.
Status lymphaticus, 826; sudden death in, 827.
Stellwag's sign, 838.
Stenocardia, 761.
Stenosis, of aortic orifice, 715; of mitral orifice, 721; of pulmonary orifice, 727, 767; of tricuspid orifice, 726.
Steppage gait, 1034.
Stercoraceous vomiting, 534.
Stercoral ulcers in colitis, 513.
Stertor, in apoplexy, 1001.
Stiff neck, 406.
Stigmata, in hysteria, 1118; in purpura, 815.
Stitch in side in pneumonia, 115; in pleurisy, 668.

Stokes-Adams syndrome, 760.
Stolidity of face in general paresis, 962.
Stomach, acute cancer of, 493.
Stomach, cancer of, 486; absence of free HCl in, 491; diagnosis from gastric ulcer and chronic gastritis, 493; hæmorrhage in, 490; vomiting in, 490.
Stomach, dilatation of, 474; tetany in, 475.
Stomach, diseases of, 463.
Stomach, atrophy of, 467; atony of, 500; chronic catarrh of, 466; erosions of, 468; foreign bodies in, 494; hæmorrhage from, 481, 495; hair tumors in, 494; neuroses of, 497; non-cancerous tumors in, 494; tuberculosis of, 319; ulcer of, 478; washing out of (lavage), 472.
Stomatitis, 441; acute, 441; aphthous, 441; epidemic, 347; fetid, 442; follicular, 441; gangrenous, 444; mercurial, 444; neurotica chronica, 443; parasitic, 443; ulcerative, 442; vesicular, 441; uræmic, 868.
Stone-cutter's phthisis, 269, 652.
Stools, of acute yellow atrophy, 552; of cholera, 179; of dysentery, 195, 197, 199; of typhoid fever, 23; in hæmatemesis, 497; of obstructive jaundice, 549.
Strabismus, 1048.
Strangulation of bowel, 531, 536.
"Strawberry" tongue in scarlet fever, 78.
Streptococci in diphtheria, 141; in empyema, 671; in endocarditis, 702; in pneumonia, 113; in peritonitis, 597; in pyæmia, 163; in rheumatic fever, 168; in scarlet fever, 77; in septicæmia, 162; in tonsillitis, 451.
Streptococcus diphtheritis, 142.
Streptococcus erysipelatos, 157.
Streptococcus pyogenes in erysipelas, 157.
Streptothrix actinomyces, 235.
Strictures and tumors of the bowel, 533.
Stricture of bile-duct, 560.
Stricture of colon, cancerous, 533.
Stricture of intestine, 533; after dysentery, 200, 533; after tuberculous ulcer, 319.
Stricture of œsophagus, 460.
Stricture of pylorus, 494.
Strongyloides intestinalis, 364.
Strongylus, 359.
Strumitis, 836.
Stuttering in mouth-breathers, 456.
Styrian peasants, arsenical habit in, 391.
Subclavian artery, murmur in and throbbing of, in phthisis, 308, 309.
Subphrenic peritonitis, 600.
Subsultus tendinum in typhoid fever, 29.
Succussion, Hippocratic, 683.
Succussion splash in dilated stomach, 476.
Sudamina in typhoid fever, 17.
Sudden death, in angina pectoris, 762; in aortic insufficiency, 712; in coronary artery disease, 747; in enlarged thymus, 844; in pleural effusions, 671; in status lymphaticus, 827; in typhoid fever, 40.
Sudoral form of typhoid fever, 18.
Sugar in the urine, 423.
Sulphocyanides in excess in saliva in rheumatism, 170.
Sun-stroke, 395; after-effects of, 397.
Suppression of urine, 859.
Suppurative nephritis, 887.
Suppurative pylephlebitis, 555.
Suppurative tonsillitis, 452.
Suprarenal bodies, diseases of, 828; hæmorrhage into, 832; tuberculosis of, 832; tumors of, 832.
Surgical kidney, 887.
Suspension in compression paraplegia, 972.
Sweating in acute rheumatism, 169; in ague, 212; in diabetes, 423; in phthisis, 306; in pyæmia, 164; in typhoid fever, 18; in ulcerative endocarditis, 704; profuse, in rickets, 436; unilateral, in cervical caries, 971; unilateral, in aneurism, 782.
Sweating sickness, 346.
Sydenham's chorea, 1079.
Symmetrical gangrene, 1138.
Sympathetic ganglia, in *Addison's* disease, 829.
Sympathetic nerve fibres (see Vaso-motor).
Symptomatic parotitis, 447.
Syncope, fatal, in diphtheria, 151; in cardiac disease, 712, 750; in phthisis, 317; in pleural effusion, 671.
Syncope, local, 1137.
Synovial rheumatism (see Gonorrhœal Rheumatism), 256.
Synovitis, gonorrhœal, 257.
Synovitis, symmetrical, in congenital syphilis, 244.
Syphilides, macular, 240; papular, 240; pustular, 240; squamous, 241; the late, 241.
Syphilis, 238; accidental infection in, 238; acquired, 240; amyloid degeneration in, 242; bone lesions of, 244; congenital, 242; early nerve lesions in, 245; gummata in, 239; hereditary transmission of, 238; modes of infection in, 238; of brain and cord, 244, 1020; of circulatory system, 250; of digestive tract, 249; of liver, 248; of lung, 247; orchitis in, 251; primary stage of, 240; prophylaxis of, 252; renal, 250; secondary stage of, 240; tertiary stage of, 241; visceral, 244.
Syphilis and dementia paralytica, 242, 246, 961.
Syphilis and locomotor ataxia, 242, 920.
Syphilis hæmorrhagica neonatorum, 243, 818.
Syphilitic arteritis, 245.
Syphilitic fever, 240.
Syphilitic nephritis, 250.

Syphilitic phthisis, 247.
Syringo-myelia, 975.

Tabes, diabetic, 426.
Tabes dorsalis (see Locomotor Ataxia), 920; in chronic ergotism, 394.
Tabes dorsalis spasmodique, 937.
Tabes mesenterica, 283.
Tache cérébrale, 18, 278.
Taches bleuâtres, 17, 377.
Tachycardia, 758, 838; neurasthenic, 1126; paroxysmal, 758.
Tactile fremitis, in emphysema, 658; in pneumonia, 119; in pleural effusion, 668; in pneumothorax, 682; in pulmonary tuberculosis, 307; at right apex, 307.
Tænia echinococcus, 368, 370.
Tænia elliptica, T. cucumerina, T. flavopunctata, T. nana, T. Madagascariensis, T. confusa, 366.
Tænia saginata or mediocanellata, 366.
Tænia solium, 365.
Tape-worms, 365; treatment of, 367.
Taste, disturbances of, 1060; tests for sense of, 1060.
Tea, neuritis caused by, 1035.
Techomyza fusca, 379.
Teeth, actinomyces in, 236; looseness of, in scurvy, 823; effects of stomatitis on, 445; erosion of, 445; *Hutchinson's*, 243, 445; of infantile stomatitis, 445.
Teichopsia, 1102.
Telegrapher's cramp, 1108.
Temperature sense, loss of, in syringo-myelia, 975; in *Morvan's* disease, 975.
Temperature, subnormal, in acute alcoholism, 380; in acute tuberculosis, 274; in apoplexy, 1001; in heat exhaustion, 395; in malaria, 209, 215; in pulmonary tuberculosis, 306; in tuberculous meningitis, 279; in uræmia, 866.
Temporal lobe, tumors of, 1022.
Temporo-sphenoidal lobe, centre for hearing in, 1056.
Tender points in neuralgia, 1104; in neurasthenia, 1123.
Tender toes, in typhoid fever, 30.
Tendon-reflexes (see Reflexes).
Terminal infections, 165.
Tertian ague, 212.
Testes, tuberculosis of, 326; syphilis of, 251 (see also Orchitis).
Tetanus, 230; bacillus of, 231; neonatorum, 230.
Tetanus, cephalic, 232.
Tetany, 1109; after thyroidectomy, 1110; epidemic or rheumatic, 1109; in dilatation of the stomach, 475, 1110; in myxœdema, 1110; in typhoid fever, 30.
Tetrodon, poisoning by, 394.
Therapeutic test in syphilis, 251.
Therapy, serum, in plague, 193.
Thermic fever, 395.
Thermic sense, loss of, in syringo-myelia, 975.
Third nerve, diseases of, 1045.
Third nerve, recurring paralysis of, 1046; signs of paralysis of, 1046.
Thomsen's disease, 1149.
Thoracic duct, tuberculosis of, 274.
Thorax, deformity of, in mouth-breathers, 455; in rickets, 436.
Thorax in emphysema, 658; in phthisis, 268, 306.
Thorn-headed worms, 365.
Thornwaldt's disease, 457.
Thread-worm, 353.
Throbbing aorta, 786, 1126.
Thrombi in heart, 723; in diphtheria, 145; in pneumonia, 114.
Thrombi in veins in typhoid fever, 21.
Thrombi, marantic, 1015.
Thrombosis of cerebral arteries, 1008; of cerebral sinuses, 1015; of cerebral veins, 1015; of portal vein, 554.
Thrush, 443.
Thymic asthma, 618, 844.
Thymus gland, diseases of, 843; tumors of, 845; persistence of, 844; enlargement of, 844; sudden death in, 844.
Thymus gland, in acromegaly, 1143; and exophthalmic goitre, 845.
Thyroid abscess, 836.
Thyroid extract, administration of, 843, 1111.
Thyroid gland, aberrant or accessory tumors of, 836; abscess of, 836; absence of, in cretins, 840; adenomata of, 836; cancer of, 836; in exophthalmic goitre, 838; in goitre, 836; in myxœdema, 842; sarcoma of, 836; tumors of, 836.
Thyroid gland, diseases of, 835.
Thyroidism, 843.
Tic convulsif, 1055, 1089.
Tic douloureux, 1105.
Ticks, 376.
Tinnitus aurium, 1057.
Tintement métallique, 738.
Tobacco, influence of, on the heart, 764.
Tongue, atrophy of, 1066; eczema of, 445; geographical, 445; in bulbar paralysis, 932; spasm of, 1067; tuberculosis of, 318; unilateral hemiatrophy of, 1067.
Tongue, tremor of, in general paresis, 962; ulcer of frænum in whooping-cough, 93.
Tonsillitis, 451; acute, 451; albuminuria in, 452; endocarditis in, 452; in the newly married, 451.
Tonsillitis, chronic, 454; follicular, 451; lacunar, 451; suppurative, 452; and rheumatism, 451.
Tonsils, abscess of, 452; calculi of, 456; cheesy masses in, 456; enlarged, 454; tuberculosis of, 318.

Tonsils, diseases of, 451.
Tophi, 411.
Topical diagnosis, spinal, 964; cerebral, 979.
Toronto General Hospital, statistics of typhoid fever at, 3.
Torticollis, 406, 1064; congenital, 1064; facial asymmetry in, 1064; spasmodic, 1065.
Toxic gastritis, 465.
Toxines, in septicæmia, 161.
Tracheal tugging, 780.
Traction aneurism, 777.
Trance in hysteria, 1113, 1119.
Traube's semilunar space, 669.
Trauma as a factor, in delirium tremens, 382; in neurasthenia, 1132; in pneumonia, 109; in tuberculosis, 270.
Trematodes, diseases caused by, 351.
Trembles in cattle, 344.
Tremor, alcoholic, 381, 1079; in Graves' disease, 839; hereditary, 1079; hysterical, 1079, 1115; in exophthalmic goitre, 839; lead, 389; in paralysis agitans, 1077; *Rendu's* type of, 1115; senile, 1079; simple, 1079; toxic, 1079; volitional, in insular sclerosis, 959.
Trichina spiralis, 354; distribution of, 355; statistics of, in American hogs, 355; in Germany, 355; modes of infection, 356.
Trichiniasis, 354; epidemics of, 356; prophylaxis of, 359.
Trichocephalus dispar, 364.
Trichomonas vaginalis, 351; T. hominis, 351.
Trichter-brust, 307, 455.
Tricuspid orifice, stenosis of, 726.
Tricuspid valve, disease of, 725; insufficiency of, 725.
Trigeminus (see Fifth Nerve).
Trismus, neonatorum, 230; hysterical, 1114.
Trommer's test, 423.
Trophic disorders, 1137.
Tropical dysentery, 195.
Trousseau's symptom, in tetany, 1110.
Tubal pregnancy, ruptured, simulating peritonitis, 600.
Tubercle bacilli, 259, 301.
Tubercle, diffuse infiltrated, 272; miliary, 270, 295; changes in, 271; structure of, 270; nodular, 270.
Tubercles, miliary, in chronic phthisis, 295.
Tubercula dolorosa, 1037.
Tuberculin, 261; test, 258; treatment, 335.
Tuberculosis, acute, 273; general or typhoid form, 274; meningeal form, 276; pulmonary form, 275.
Tuberculosis, 258; bacillus of, 259, 301; changes produced by bacillus, 270; chronic miliary, 295; of circulatory system, 327; conditions influencing infection, 267; congenital, 262; dietetic treatment of, 335; distribution of the tubercles in, 270; duration of pulmonary form of, 329; hereditary transmission of, 262; individual prophylaxis in, 330; infection by meat, 267; infection by milk, 267; infection by inhalation, 265; inoculation of, 264; in infants, 316; in old age, 316; medicinal treatment of, 336; modes of death in pulmonary, 317; modes of infection in, 262; natural or spontaneous, cure of, 331; of alimentary canal, 317; of brain and cord, 321; of Fallopian tubes, 326; of genito-urinary system, 322; of kidneys, 324; of liver, 320; of lymphatic system, 280; of mammary gland, 327; of ovaries, 326; of pericardium, 285; of peritonæum, 286; of pleura, 284; of prostate, 326; of serous membranes, 284; of testes, 326; of ureters and bladder, 325; of uterus, 326; of vesiculæ seminales, 326; pregnancy, influence of, in, 329; prophylaxis in, 330; pseudo-, 262; pulmonary, 289; and typhoid fever, 33; and valvular disease of heart, 316.
Tufnell's treatment of aneurism, 784.
Tumors of brain, 1020.
Tunnel anæmia, 360.
Twists and knots in the bowel, 533.
Tympanites, in intestinal obstruction, 535; in peritonitis, 598; in tuberculous peritonitis, 287; in typhoid fever, 25; as a cause of sudden heart-failure, 545.
Typhlitis, 519.
Typhoid fever, 1; abortive form, 34; afebrile, 17, 35; ambulatory form, 14, 34; anæmia in, 19; and tuberculosis, 49; bacillus of, 3; chills in, 17; circulatory system in, 19; diabetes in, 33; diarrhœa in, 23; digestive system in, 22; *Ehrlich's* reaction in, 31; erysipelas in, 33; grave form of, 34; hæmorrhage in, 10; hæmorrhagic, 35; historical note on, 1; in the aged, 35; in children, 35; in the fœtus, 36; in pregnancy, 35; laparotomy in, 48; liver in, 11, 27; Maidstone epidemic of, 5; meteorism in, 25; mild form, 34; modes of conveyance of, 5; nervous system in, 12, 28; noma in, 33, 35; osseous system in, 32; oysters and, 6; parotitis in, 23; perforation of bowel in, 10, 26; peritonitis in, 26, 47; post-typhoid elevations of temperature in, 16; prognosis of, 40; prophylaxis of, 41; pyuria in, 11, 32; relapses in, 36; renal system in, 31; respiratory system in, 27; serum therapy in, 47; skin rashes in, 17; spleen in, 26; tender toes in, 30; tetany in, 30; varieties of, 33; *Widal's* reaction in typhoid fever, 38.
Typhoid gangrene, 12, 22.
Typhoid septicæmia, 33.
Typhoid spine, 32.
Typhoid state in obstructive jaundice, 550; in acute yellow atrophy, 552.
Typho-malarial fever, so called, 39, 214.
Typhotoxin, 8.

Typhus fever, 49; complications and sequelæ of, 52.
Typhus siderans, 52.
Tyrosin, 552.
Tyrotoxicon, 393.
Tyrotoxismus, 393.

Ulcer, cancerous, of intestine, 513; gastric, 478; of duodenum, 478; of bowel in dysentery, 194, 196, 199; in typhoid fever, 9.
Ulcer of mouth, 442; in the new-born, 443; in nursing women, 443; of palate in infants, 443.
Ulcer, peptic, 478; perforating, of foot, in tabes, 925; in diabetes, 425.
Ulcerative endocarditis, 699.
Ulcers, *Parrot's*, 443.
Ulnar nerve, affections of, 1071.
Uncinaria duodenalis, 359.
Unconsciousness (see Coma).
Undulant fever, 219.
Uræmia, 865; cerebral manifestations of, 866; coma in, 867; convulsions in, 866; diagnosis from apoplexy, 868; dyspnœa in, 867; headache in, 867; in Bright's disease, 884; latent, 851; local palsies in, 867; œdema of brain in, 997; stomatitis in, 868; theories of, 865.
Urate (lithate) of soda in gout, 408.
Urates in the urine, 860.
Urates (lithates), amorphous, 860.
Ureter, blocking of, 850; mucous cysts of, 350; obstructed by calculi, 893; psorospermiasis of, 350; tuberculosis of, 325.
Urethritis, gouty, 415.
Uric acid, calculus, 892; deposition of, 860; in gout, 408; in urine, 860; "showers," 415.
Uric-acid diathesis (see Lithæmia), 860.
Uric-acid headache, 415.
Uric-acid theory of gout, 408.
Urinary calculi, 892.
Urine, anomalies of the secretion of, 850.
Urine, density of, in acute Bright's disease, 870; in chronic Bright's disease, 880; in diabetes, 423; in diabetes insipidus, 433.
Urine, hæmoglobin in, 852.
Urine, in acute yellow atrophy of liver, 552; in grave anæmia, 799; in cholera, 179; in diabetes insipidus, 433; in diabetes mellitus, 423; in diphtheria, 150; in erysipelas, 159; in gout, 411, 413, 415; in jaundice, 549; in melanotic sarcoma, 863; in pneumonia, 122; in acute pulmonary tuberculosis, 312; in typhoid fever, 31; oxalates in, 861; pus in, 858.
Urine, quantity of, in chronic Bright's disease, 880; in diabetes insipidus, 433; in diabetes mellitus, 423; in intestinal obstruction, 535.
Urine, retention of, in typhoid fever, 31.
Urine, suppression of, 850; treatment of, 851; in cholera, 179; in acute nephritis, 870; in scarlet fever, 80; in acute intestinal obstruction, 535; obstructive suppression, 894.
Urine, tests for albumin in, 856; biliary pigment in, 549; blood in, 852; albumoses in, 857; peptones in, 857.
Urobilin, increase of, in pernicious anæmia, 799.
Uro-genital tuberculosis, 322.
Urticaria, after tapping of hydatid cysts, 372; epidemica, 379; giant form (see Neurotic Œdema), 1141; with purpura, 815; in smallpox, 60; in typhoid fever, 18.
Uterus, tuberculosis of, 326.
Uvula, œdema of, 448; infarction of, 448, 816.

Vaccination, 68; mark, 70; technique of, 73; rashes, 71; ulcers, 71; value of, 73.
Vaccine, antityphoid, 42.
Vaccine lymph, choice of, 72.
Vaccinia, 68; bacteriology of, 70; generalized, 71.
Vaccino-syphilis, 71.
Vagabond's discoloration, 377, 831.
Valvular disease of heart, 707; and tuberculosis, 316.
Varicella, 74; hæmorrhagic, 75.
Varicella bullosa, 75; escharotica, 75.
Varices, œsophageal, in cirrhosis of liver, 459.
Variola, 56; hæmorrhagica, 59, 62, 63; vera, 59.
Variola hæmorrhagica pustulosa, 62, 63.
Variola sine eruptione, 64.
Varioloid, 59, 63.
Vaso-motor disorders, 1137.
Vaso-motor disturbances in caries, 971; in chronic pleurisy, 680; in exophthalmic goitre, 839; in hemicrania, 1103; in myelitis, 977; in neuralgia, 1104.
Veins, cerebral, thrombosis in, 1015; diastolic collapse of, 697; pulsation in, 311, 1084, 1126; sclerosis of, 773.
Vena cava, inferior, twist in, 668.
Vena cava, superior, perforation of, by aneurism, 778, 788.
Venereal disease, 238.
Venesection (see Bloodletting).
Venous pulse, 311, 1084, 1126.
Ventricles of brain, dilatation of (hydrocephalus), 1028; puncture of, 1030.
Ventricular hæmorrhage, 999.
Verruca necrogenica, 264.
Vertebræ, caries of, 970; cervical, caries of, 971.
Vertebral artery, obstruction of, 1010.
Vertigo, auditory, 1058; cerebellar, 986; in arterio-sclerosis, 775; in brain tumor, 1021; gastric, 469; labyrinthine, 1058; endemic paralytic, 1059.
Vesiculæ seminales, tuberculosis of, 326.
Vicarious, epistaxis, 614; hæmoptysis, 637.
Virus fixe, 229.
Visceroptosis, 541.

Vitiligoidea, 549.
Vocal fremitus, 119, 668; resonance, 120, 670.
Voice (see SPEECH).
Voice, alteration of, in mouth-breathers, 456.
Volitional tremor, 959.
Volvulus, 533, 537.
Vomica, 296; signs of, in phthisis, 309.
Vomit, black, 186; coffee-ground, 490.
Vomiting, in *Addison's* disease, 880; in *Bright's* disease, 881; in cerebral abscess, 1026; in cerebral tumor, 1021; in chronic obstruction of intestines, 535; in chronic ulcerative phthisis, 311; in gall-stone colic, 564; in gastric cancer, 490; in gastric ulcer, 481; in acute obstruction of intestines, 534; in tuberculous meningitis, 278; in migraine, 1103; in peritonitis, 598; in small-pox, 59; nervous, 499; primary periodic, 499; stercoraceous, 534; uræmic, 867.
Vulvitis, ulcerative, in measles, 87.

Wall-paper, poisoning by arsenic in, 390.
Wart-pox, 63.
Warts, post-mortem, 264.
Washing out stomach, 472, 477.
Water-hammer pulse, 714.
Water, infection by, in diphtheria, 138; in cholera, 177; in typhoid fever, 5.
"Water on the brain," 276.
Weber, syndrome of, 279, 1004, 1023.
Weil's disease, 344.
Werlhoff's disease, 816.
Wernicke's hemiopic pupillary inaction, 1044.
Wet-pack, 84.
Whip-worm, 364.
White softening of brain, 1009.
White thrombi in heart, 723.
Whooping-cough, 92.
Winckel's disease (see EPIDEMIC HÆMOGLOBINURIA OF THE NEW-BORN), 243, 818, 853.
"Winged scapulæ," 307.
Wintrich's sign, 309.
Woillez, maladie de, 634.
Wool-sorter's disease, 224, 226.
Word-blindness, 989.
Word-deafness, 989.
Word-dumbness, 992.
Wormian bones in hydrocephalus, 1029.
Worms (see PARASITES).
Wounds of the heart, 754.
Wrist-drop, 1071; in lead-poisoning, 388.
Writer's cramp, 1107.
Wryneck, 1064; spasmodic, 1065.

Xanthelasma, 549.
Xanthine calculi, 892.
Xanthomata, 425, 549, 565.
Xanthopsia, 353.
Xerostomia, 447.

Yellow fever, 182; bacteriology of, 183; epidemics of, 182.
Yellow softening of brain, 1009.
Yellow vision, 353.
Yeo's dietary in obesity, 440.

Zona, 1106.

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